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Applied Science Associates, Inc.
Valencia, PA

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This handbook explains the effects of various air pollutants on vegetation and how to diagnos the suspected injuries. It discusses the structure and function of plants in relation to air pollution injury and the different pollutants: photochemical oxidants, sulfur dioxide, fluorides, and minor pollutants. It also explains the interactions between pollutants and pollutants and pathogens, discusses mimicking symptoms, and identifies the sources and meteorological aspects of air pollution and the effects of topography on dispersion. The basic procedure and routine for diagnosis of suspected air pollution injury to vegetation is outlines. Charts, glossary and index.

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OFFICE OF AIR AND WASTE MANAGEMENT

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BY
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FOREWORD

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TABLE OF CONTENTS

Page

Chapter 1. Introduction to Diagnosis of Air Pollution Damage to Vegetation 1-1

Selected References 1-6

Chapter 2. Structure and Function of Plants in Relation to Air Pollution Injury 2-1

Introduction 2-1
Plant Cell Ultrastructure 2-1
Plant Organs 2-3
Roots 2-3
Stems 2-6
Leaves 2-8
Flowers, Fruits and Seeds 2-12

Selected References 2-13

Chapter 3. The Photochemical Oxidants 3-1

Introduction 3-1

SECTION 1. OZONE 3-2
Sources 3-2
Atmospheric Chemistry 3-4
Monitoring Data 3-7
Symptomatology 3-8
Factors Affecting Plant Response 3-11
Injury Threshold Doses 3-12
Air Quality Standards 3-13
Relative Sensitivity of Plants to Ozone 3-14
Leaf Tissue Analysis 3-16

SECTION 2. PAN 3-17
Introduction 3-17
Symptomatology 3-17
Factors Affecting Plant Response 3-19
Injury Threshold Doses 3-19
Air Quality Standards 3-19
Relative Sensitivity of Plants to PAN 3-19
Leaf Tissue Analysis 3-21

SECTION 3. OXIDES OF NITROGEN 3-22
Introduction 3-22
Sources 3-22
Atmospheric Chemistry 3-22
Monitoring Data 3-23

Page

  Symptomatology 3-26
  Factors Affecting Plant Response 3-27
  Injury Threshold Doses 3-28
  Air Quality Standards 3-29
  Relative Sensitivity of Plants to NO_2 3-29
  Leaf Tissue Analysis 3-31

Selected References 3-31

Chapter 4. Sulfur Dioxide 4-1

Introduction 4-1
Sources 4-1
Atmospheric Chemistry 4-2
Monitoring Data 4-2
Symptomatology 4-4
Factors Affecting Plant Response 4-6
Injury Threshold Doses 4-8
Air Quality Standards 4-10
Relative Sensitivity of Plants to SO_2 4-10
Leaf Tissue Analysis 4-11

Selected References 4-11

Chapter 5. Fluorides 5-1

Introduction 5-1
Sources 5-1
Atmospheric Chemistry 5-3
Monitoring Data 5-4
Symptomatology 5-4
Factors Affecting Plant Response 5-10
Injury Threshold Doses 5-13
Air Quality Standards 5-17
Relative Sensitivity of Plants to HF 5-17
Leaf Tissue Analysis 5-17

Selected References 5-18

Chapter 6. Minor Pollutants 6-1

Introduction 6-1

  SECTION 1. CHLORINE 6-2
  Introduction 6-2
  Sources 6-2
  Atmospheric Chemistry 6-2
  Monitoring Data 6-3
  Symptomatology 6-3

Page

Factors Affecting Plant Response 6-4
Injury Threshold Doses 6-4
Air Quality Standards 6-4
Relative Sensitivity of Plants to Chlorine 6-4
Leaf Tissue Analysis 6-5

SECTION 2. HYDROGEN CHLORIDE 6-6
Introduction 6-6
Sources 6-6
Atmospheric Chemistry 6-6
Monitoring Data 6-7
Symptomatology 6-7
Factors Affecting Plant Response 6-7
Injury Threshold 6-8
Air Quality Standards 6-8
Relative Sensitivity of Plants to Hydrogen Chloride 6-8
Leaf Tissue Analysis 6-9

SECTION 3. ETHYLENE 6-10
Introduction 6-10
Sources 6-10
Atmospheric Chemistry 6-10
Monitoring Data 6-11
Symptomatology 6-11
Factors Affecting Plant Response 6-13
Air Quality Standards 6-13
Relative Sensitivity of Plants to Ethylene 6-14
Leaf Tissue Analysis 6-14

SECTION 4. AMMONIA 6-15
Introduction 6-15
Sources 6-15
Atmospheric Chemistry 6-15
Monitoring Data 6-15
Symptomatology 6-16
Factors Affecting Plant Response 6-16
Injury Threshold 6-17
Air Quality Standards 6-17
Relative Sensitivity of Plants to Ammonia 6-17
Leaf Tissue Analysis 6-17

SECTION 5. HYDROGEN SULFIDE 6-18
Introduction 6-18
Sources 6-18
Atmospheric Chemistry 6-18
Monitoring Data 6-18
Symptomatology 6-18

Page

Factors Affecting Plant Response 6-19
Injury Threshold 6-19
Air Quality Standards 6-19
Relative Sensitivity of Plants to Hydrogen Sulfide 6-19
Leaf Tissue Analysis 6-19

SECTION 6. CARBON MONOXIDE 6-21
Introduction 6-21
Sources 6-21
Symptomatology 6-21

SECTION 7. HEAVY METALS 6-23
Introduction 6-23
Lead 6-23
Zinc, Cadmium and Copper 6-24
Mercury Vapors 6-24
Relative Sensitivity of Plants to Mercury Vapors 6-25
Leaf Tissue Analysis 6-26

SECTION 8. PARTICULATE MATTER 6-27
Introduction 6-27
Sources 6-27
Atmospheric Chemistry 6-27
Symptomatology 6-27
Air Quality Standards 6-28

SECTION 9. PESTICIDES 6-29
Introduction 6-29
Sources 6-29
Symptomatology 6-30
Relative Sensitivity of Plants to Pesticides 6-30
Leaf Tissue Analysis 6-30

Selected References 6-32

Chapter 7. Interactions Between Pollutants and Between Pollutants and Pathogens 7-1

Pollutant – Pollutant Interactions 7-1
Pollutant – Pathogen Interactions 7-5

Selected References 7-6

Chapter 8. Mimicking Symptoms 8-1

Abiotic Agents 8-1
Biotic Agents 8-5
Teratogenic Agents 8-7
Summary 8-7

Selected References 8-8

Page

Chapter 9. Meteorology and Air Pollution Injury to Vegetation 9-1

Sources of Air Pollution 9-1
Meteorological Aspects of Air Pollution 9-2
Effects of Topography on Dispersion 9-7

Chapter 10. Diagnosis of Suspected Air Pollution Injury to Vegetation 10-1

Diagnosis – Basic Procedure 10-1
Diagnosis – Systematic Approach 10-5
Diagnostic Routine 10-8

Appendix A. Conversion Factors A-1

Appendix B. National Primary and Secondary Air Quality Standards B-1

Appendix C. Composite Sensitivity Chart and Scientific Names of Plants C-1

Appendix D. Bio-Indicators D-1

Appendix E. Glossary of Air Pollution and Botanical Terms E-1

Index Index-1

LIST OF TABLES

Table No.		*Page*
3-1	Data Showing Long-term Trends in Oxidant Concentrations Recorded at CAMP Sites from 1964 to 1967 in Four Large Urban Areas	3-7
3-2	Threshold Doses of Ozone Needed to Cause Injury to Plants Grown Under Sensitive Conditions	3-13
3-3	Relative Sensitivity of Various Plants to Ozone	3-14
3-4	Relative Sensitivity of Various Plants to PAN	3-20
3-5	Relative Sensitivity of Various Plants to NO_2	3-29
4-1	Estimated Total Nationwide SO_2 Emissions Levels, 1940-1970	4-2
4-2	Nationwide Estimates of SO_2 Emissions from Various Sources During 1970	4-3
4-3	Sulfur Dioxide Annual Average Concentration of Four Air Quality Control Regions for 1964 and 1971, in $\mu g/m^3$	4-3
4-4	Relative Sensitivity of Various Plants to SO_2	4-9
5-1	Estimated Total Fluoride Emissions from Major Industrial Sources in the United States in 1968	5-3
5-2	Relative Sensitivity of Various Plants to F	5-15
6-1	Threshold Concentrations for Acute Chlorine Injury	6-4
6-2	Relative Sensitivity of Various Plants to Chlorine	6-5
6-3	Relative Sensitivity of Various Plants to Hydrogen Chloride	6-8
6-4	Dose Response of Plants to Ethylene	6-13
6-5	Relative Sensitivity of Various Plants to Ethylene	6-14
6-6	Relative Sensitivity of Various Plants to Ammonia	6-17
6-7	Relative Sensitivity of Various Plants to Hydrogen Sulfide	6-20
6-8	Relative Sensitivity of Various Plants to Mercury Vapors	6-25
6-9	Relative Sensitivity of Various Plants to 2,4-D	6-31
7-1	Selected Examples of Effects of Sulfur Dioxide and Ozone Mixtures on Foliar Injury	7-2
7-2	Selected Examples of Effects of Sulfur Dioxide and Nitrogen Dioxide Mixtures on Foliar Injury	7-3

Preceding page blank

Table No. *Page*

7-3 Selected Examples of Effects of Sulfur Dioxide and Hydrogen Fluoride Mixtures on Foliar Injury 7-4

7-4 Selected Examples of Pollutant-Pathogen Interaction 7-5

8-1 List of Agents That Can Induce Plant Diseases 8-1

8-2 Cross-Reference of Symptoms Which May be Confused for Air Pollution Injury by the Layman 8-7

D-1 Distribution of Epiphytic Lichens in Various IAP Zones D-2

D-2 List of Common Bio-indicators and Pollutant(s) to Which They Are Sensitive D-4

LIST OF FIGURES

Figure No. *Page*

3-1 Atmospheric NO_2 Photolytic Cycle 3-3
3-2 Photolytic Cycle with Hydrocarbons 3-3
3-3 Monthly Variation of Mean Daily Maximum One-Hour Average Oxidant Concentrations for Three Selected Cities ... 3-4
3-4 Average Daily One-Hour Concentrations of Selected Pollutants in Los Angeles, July 19, 1965 3-5
3-5 Diurnal Variations of Mean Hourly Average Oxidant Concentrations in Philadelphia, August 6-8, 1966 3-6
3-6 Diurnal Variation of Mean Hourly Average Oxidant Concentrations in Los Angeles and St. Louis 3-6
3-7 Weekday and Weekend One-Hour Average NO Levels in Chicago, 1962 through 1964 3-24
3-8 Average and Standard Deviation of Daily Maximum Oxides of Nitrogen and Nitrogen Dioxide Concentrations for Seven Locations in Los Angeles Basin, 1962 3-24
3-9 Trend Lines for NO_x Annual Averages in Five CAMP Cities 3-25
3-10 Approximate Thresholds for Death, Leaf Injury, and No Effect, as Related to NO_2 Concentration and Duration of Exposure 3-28
5-1 Composite Picture of Environmental Fluoride 5-2
5-2 The Relationship of Concentration of Atmospheric Fluoride and Duration of Exposure to the Threshold for Foliar Symptoms for Sensitive, Intermediate, and Tolerant Species of Plants 5-14
6-1 Trend Lines for CO Annual Averages in Five CAMP Cities ... 6-22
9-1 Temperature Variations with Altitude 9-4
9-2 Temperature Variation with Altitude Illustrating a Temperature Inversion Based at the Earth's Surface 9-4
9-3 Temperature Profile Illustrating Two Inversion Layers 9-5
9-4 Conditions of Figure 9-3 in a Different Perspective 9-6
10-1 Topographical Features of the Hypothetical Example 10-6
10-2 The One Pathway Followed to Solve a Particular Problem ... 10-9

LIST OF PHOTOGRAPHS

Plate No.		Page
2-1	Leaf Cells of *Spartina alterniflora* (Smooth Cord Grass) Showing Cell Organelles	2-14
2-2	Portion of Root Meristem Cell of Chrysanthemum Showing Cell Organelles	2-15
2-3	Section Through Chloroplasts of a Chrysanthemum Leaf Cell Showing Details of Grana and Lamella	2-16
2-4	Cross Section of a *Ranunculus sp.* (Buttercup) Root	2-17
2-5	Cross Section Showing the Stele Area of a *Ranunculus sp.* (Buttercup) Root	2-18
2-6	Longitudinal Section Through the Apical Meristem of *Strophostyles helvola* (Beach Pea) Root Showing Area Where Primary Growth Occurs	2-19
2-7	Cross Section of the Root of *Nicotiana tobaccum* (Tobacco) Showing Secondary Growth and Lateral Root Formation	2-20
2-8	Cross Section of the Herbaceous Dicot Stem of *Medicago sativa* (Alfalfa)	2-21
2-9	Cross Section of the Woody Dicot Stem of *Liriodendron tulipifera* (Tulip Poplar)	2-22
2-10	Cross Section of the Gymnosperm Stem of *Pinus sp.* (Pine)	2-23
2-11	Cross Section of the Outer Area of the Gymnosperm Stem of *Pinus sp.* (Pine) Showing Sites of Secondary Growth	2-24
2-12	Cross Section of the Monocot Stem of *Zea mays* (Corn)	2-25
2-13	Longitudinal Section of the Shoot Tip of *Spartina alterniflora* (Smooth Cord Grass) Showing the Apical Meristem, Young Leaves and Intercalary Meristems	2-26
2-14	Cross Section of the Broad-leaved Leaf of *Glycine max* (Soybean)	2-27
2-15	Cross Section of the Narrow-leaved Leaf of *Zea mays* (Corn)	2-28
2-16	Cross Section of the Needle-like Leaf of *Pinus sp.* (Pine)	2-29
2-17	Surface View of a Stoma of *Zebrina sp.* (Wandering Jew)	2-30
2-18	Cross Section of the Outer Bark of *Liriodendron tulipifera* (Tulip Poplar) Showing a Lenticel	2-31

Preceding page blank

Plate No. *Page*

3-1 Example of Initial Water-Soaked Appearance on Broccoli Immediately after Exposure. This Plant was Fumigated with 10 pphm O_3 and 0.5 ppm SO_2 3-33
3-2 Bel W-3 Tobacco Injured by Ambient Oxidant in St. Louis, MO, Showing Both Fleck and Bifacial Necrosis 3-33
3-3 Ozone Injury (Flecking) to White Ash 3-34
3-4 Ozone Injury to Swiss Chard 3-34
3-5 Ozone Injury to Tobacco 3-35
3-6 Ozone Injury to Radish 3-35
3-7 Ozone Injury to Radish and Subsequent Reduced Yield on Right 3-36
3-8 Cucumber Injured by Ambient Oxidant Near Cincinnati, Ohio 3-36
3-9 Oxidant Injury to Squash 3-37
3-10 Tomato Leaflet Showing Oxidant Injury (Probably Ozone) 3-37
3-11 Safflower Leaf Showing Oxidant Injury (Probably Ozone) 3-38
3-12 Soybean Leaves Showing Various Stages of Oxidant Injury (Normal Leaf in Center; Leaflet on Right is Secondary Response to Oxidants) 3-38
3-13 Tempo Bean Leaves Showing Oxidant Injury (Probably Ozone) 3-39
3-14 Bronzing of White Bean Leaves Caused by Oxidant (Probably Ozone) 3-39
3-15 Severe Ozone Injury and Bifacial Necrosis of Browallia Leaves 3-40
3-16 Severe Grape Stipple Caused by Ozone 3-40
3-17 Pinto Bean Injured by Ambient Oxidant Near Cincinnati, Ohio, Showing Stipple Symptom 3-41
3-18 Ozone Stipple of White Ash as Observed in the Field 3-41
3-19 Bluegrass Injured by Ambient Oxidant in the Los Angeles Basin 3-42
3-20 Severe Oxidant Injury to Ponderosa Pine 3-43
3-21 Chronic Ozone Injury of Ponderosa Pine. Note Difference in Tolerance Between Trees and Tufted Appearance of Foliage 3-44

Plate No.		Page
3-22	Tipburn of Onion Caused by Ozone	3-45
3-23	Eastern White Pine Displaying Oxidant Injury (Probably Ozone)	3-45
3-24	Comparison of Ozone and PAN Injury on Alfalfa. Upper row: Ozone Injury; Lower row: PAN Injury	3-46
3-25	PAN Injury to a Bean Leaflet. Left, Upper Leaf Surface; Right, Lower Leaf Surface Showing Silvering or Glazing Due to PAN	3-46
3-26	Young Primary Leaf of Pinto Bean Injured by PAN After Exposure to Irradiated Auto Exhaust	3-47
3-27	Escarole Showing Oxidant Injury (Probably Due to PAN)	3-47
3-28	PAN (Ambient) Injury to Swiss Chard	3-48
3-29	Petunia with PAN or Ozone Injury	3-48
3-30	PAN Injury to Oats	3-49
3-31	Injury on Broccoli One Week After Exposure to NO_2. Leaves Show Some Recovery from Initial Water-Soaking and also Some Marginal and Interveinal Necrosis	3-49
3-32	Marginal and Interveinal Injury on Periwinkle Fumigated with 19.3 ppm of NO_2 for 0.5 Hours	3-50
3-33	Marginal and Interveinal Injury on Cherry Belle Radish Fumigated with 7.7 ppm NO_2 for Four Hours	3-50
4-1	Acute Sulfur Dioxide Injury to Bracken Fern, Typically Orange-Red; Occurs on Margins of Fronds	4-12
4-2	Typical Sulfur Dioxide Injury to Fox Grape. Light Brown, Occurs Between Veins; Top Leaves Show Upper Surface; Bottom Leaves Show Undersurface	4-12
4-3	Acute Sulfur Dioxide Injury to White Birch Foliage	4-13
4-4	Acute Sulfur Dioxide Injury to Trembling Aspen Leaves. Injured Areas are Reddish-Brown and Darken with Age	4-14
4-5	Typical Acute Sulfur Dioxide Injury to the Garden Pea	4-15

Plate No.		*Page*
4-6	Corn Foliage Exhibiting Acute Sulfur Dioxide Injury (Injured Areas are Composed of White-to-Ivory Colored Collapsed Cells)	4-15
4-7	Light Acute Sulfur Dioxide Injury to Alfalfa	4-16
4-8	Severe Acute Sulfur Dioxide Injury to Alfalfa Leaves. Injury Ivory Colored, Marginal with Intercostal Spotting	4-16
4-9	Acute Sulfur Dioxide Injury on Blackberry Growing Within Five Miles of the Source	4-17
4-10	Typical Acute Sulfur Dioxide Injury to Hickory. Reddish-Brown; Frequently Extends from Near Midrib to Near Edge of Leaf	4-17
4-11	Acute Sulfur Dioxide Injury to Eggplant Leaves (Ivory-to-Light Tan)	4-18
4-12	Acute Sulfur Dioxide Injury to Quince Foliage (Colorful Brownish-Red)	4-18
4-13	Acute Sulfur Dioxide Injury to Careless Weed (Ivory-Colored; Note Little or No Injury on Young Expanding Leaves)	4-19
4-14	Chronic Sulfur Dioxide Damage to Cotton Foliage in Palm of Hand. Note Injured Areas are Chlorotic But Not Collapsed; Veins Usually Remain Green	4-19
4-15	Acute Sulfur Dioxide Injury to Sonora Wheat (Ivory-Colored)	4-20
4-16	Suspected Sulfur Dioxide Injury on Virginia Pine Growing 1.5 Miles from a Source	4-21
4-17	Typically Acute Sulfur Dioxide Injury to Loblolly Pine Needles	4-22
4-18	Acute Sulfur Dioxide Injury to Yellow Summer Squash. Acute Injury to all Squash and Pumpkin Plants Is White or Ivory-Colored	4-22
4-19	Acute Sulfur Dioxide Injury to Tulip. Note Ivory-Colored Injury Occurs at Leaf Tips and Margins	4-23
5-1	Citrus Foliage with the Typical Fluoride-Induced Pattern of Chlorosis	5-19
5-2	Blueberry Leaves Showing the Kind of Injury Induced by Atmospheric Fluoride	5-19
5-3	Marginal and Intercostal Necrosis of a Grape Leaf	5-20

Plate No.		Page
5-4	Hydrogen Fluoride Injury to Poplar (Field Exposure)	5-20
5-5	Oregon Grape with Symptoms Mimicking Fluoride Injury, Cause Not Known	5-21
5-6	Lamb's Quarters Showing HF-Induced Injury	5-21
5-7	Leaves of Sweet Corn Showing HF-Induced Symptoms and Varietal Differences in Tolerance. From Top (Tolerant) to Bottom (Sensitive) the Varieties are: Surecross, Golden Cross Bantam, Gold Rush, Spancross, and Marcross	5-22
5-8	Injury Due to HF on Gladiolus, Pine, and Tulip Fumigated Together	5-22
5-9	Fluoride-Induced Injury on Gladiolus Foliage	5-23
5-10	Fluoride-Induced Injury on Fir; Note Several Uninjured Needles	5-23
5-11	Slash Pine Displaying the Reddish-Brown Discoloration of Needles Injured by Fluoride	5-24
5-12	Suture Red Spot Syndrome of Peach Fruit Induced by HF on Dwarf Elberta	5-24
5-13	Fluoride Injury to Ponderosa Pine. Note That Only One Tree Exhibits Injury. This is an Illustration of the Natural Differences in Tolerance Found in the Field	5-25
6-1	Chlorine Injury to Sassafras Showing Reddish-Brown Interveinal Necrosis	6-33
6-2	Chlorine Injury to Witch Hazel Showing Necrotic Lesions Along Veins and a Portion of the Leaf Margins	6-33
6-3	Chlorine Injury to Italian Prune Showing Upper Leaf Surface Flecking	6-34
6-4	Chlorine Injury on Wild Mustard Showing Bleaching Effect	6-34
6-5	Chlorine Injury on Pine Showing Reddish-Brown Discoloration	6-35
6-6	Chlorine Injury on Larch from Chemical Plant Spill. Note Chlorotic Mottle	6-35
6-7	Hydrogen Chloride Injury to Norway Maple Showing Marginal Necrosis	6-36
6-8	Hydrogen Chloride Injury to European Black Alder Showing Interveinal Necrosis	6-36

Plate No. *Page*

6-9 Hydrogen Chloride Injury to Black Cherry Showing Leaf Spotting 6-37

6-10 Hydrogen Chloride Injury on Norway Spruce Showing Tip Necrosis 6-37

6-11 Ethylene Injury on Orchid Showing "Dry Sepal" on Flower at the Left 6-38

6-12 Azalea Fumigated with 2 ppm Ethylene for Seven Days. Note Heavy Leaf Drop and Loss of Flowers. Control Plant is on Right 6-38

6-13 Ammonia Injury on Poison Ivy Showing Blackened Appearance. . 6-39

6-14 Ammonia Injury on Cotton 6-39

6-15 Cement Dust Deposit on Fir Branch 6-40

6-16 Lime Dust Deposit on Spruce Twigs on the Right 6-40

6-17 2,4-D Injury to Oak Leaves Showing "Lacy" Appearance of Leaves 6-41

6-18 Atrazine Injury on Soybeans. Grain Sorghum was Sprayed with Atrazine During Previous Growing Season 6-41

7-1 Bel W-3 Tobacco Exhibiting Injury from Fumigation with .05 ppm O_3 and .10 ppm of SO_2. Note the Similarity to Ozone Injury 7-7

7-2 Injury to Bel W-3 Tobacco Fumigated with .05 ppm of O_3 and .50 ppm of SO_2. In This Case the Injury More Closely Resembles SO_2 Injury 7-7

7-3 Pinto Bean Fumigated with .15 ppm of O_3 and .50 ppm of SO_2. Note the Almost Complete Absence of Bleaching and the Slight Purplish Discoloration 7-8

7-4 Bleaching of Tomato Leaf as a Result of Fumigation with .05 ppm of O_3 and .25 ppm of SO_2 7-8

7-5 Lower Surface of Soybean Leaf Fumigated with .10 ppm of O_3 and .50 ppm of SO_2. Note the Difference in Symptoms from Plate 7-6 7-9

7-6 Soybean Leaf Fumigated with .10 ppm of O_3 and .50 ppm of SO_2 7-9

7-7 Bel W-3 Tobacco Fumigated with .10 ppm of SO_2 and .10 ppm of NO_2 7-10

7-8 Lower Leaf Surface of Pinto Bean Leaf Fumigated with .05 ppm of SO_2 and .10 ppm of NO_2 7-10

Plate No. *Page*

8-1 Heat Injury to Roadside Trees Caused by Street Paving 8-9

8-2 Winter Injury to Boxwood and Deodora Cedar. Many Different Agents Could Cause Similar Damage 8-10

8-3 Roadside Maples Affected by De-Icing Salts. Position of the Trees and the History of Salt Application are Keys to Proper Diagnosis 8-11

8-4 Roadside Shrubs Affected by Salt Spray from Passing Vehicles Following Use of De-Icing Salts 8-12

8-5 Injury to Oaks Caused by Grade Changes in Landscaping. Bronzing of Foliage and Leaf Drop Preceded Death 8-12

8-6 Frost Injury to White Oak. Note Similarity of Damage Symptoms to SO_2 or F-Induced Injury 8-13

8-7 Nitrogen Deficient Corn Leaves, Showing Symptoms That Could Readily Be Confirmed Through a Soil or Foliage Test 8-13

8-8 Manganese Deficient Grape Leaves Showing Symptoms Similar to Those Induced by Ozone 8-14

8-9 Miticide (Kelthane) Injury on Soybean Leaves. Note Similarity to Air Pollution Injury 8-14

8-10 Needlecast of Scotch Pine. Similar Initial Symptoms Could be Caused by SO_2, O_3, or F 8-15

8-11 Bluegrass Injured by Thrips (center); Normal Leaves (right); O_3 Injured Leaves (left) 8-15

8-12 Pine Sawfly Damage to Loblolly Pine Stand. Close Examination Would Easily Indicate This Cause Unless the Insect Was Not Present 8-16

8-13 Mite Injury to Tomato Leaflets Grown in the Field. Note Similarity to Ozone Injury 8-16

8-14 Soybean Leaves. Left, Oxidant Injury (Probably Ozone); Center, Normal Leaflet; Right, Mite Injury 8-18

8-15 Dogwood with Lacewing Injury. Note Similarity of Insect Damage to Air Pollution Injury 8-17

8-16 Eastern White Pine with Oyster Scale Insect Injury. Note Similarity to Air Pollution Injury 8-18

Chapter 1

INTRODUCTION TO DIAGNOSIS OF AIR POLLUTION DAMAGE TO VEGETATION

A Brief History

Natural sources of air pollution have been with us for a long time, but they did not achieve the status of a major social problem until the 1300's. The introduction of coal as a source of energy polluted the air of populated areas with sulfur dioxide and particulates, irritating the mucous membranes and esthetic senses of rich and poor alike.*

As technology grew, various industries added their own brands of pollutants to the air. The metallurgical industry contributed contaminants associated with metal processing such as sulfur dioxide, flouride, and heavy metals. The chemical industry donated hydrochloric acid, chlorine, ammonia, hydrogen sulfide, nitrogen oxides, and others. Each major industry has added its own particular complex of pollutants, making the problem of control progressively more difficult.

In recent decades, automobile exhaust has become a massive, widespread problem. Petroleum components in the exhaust react with atmospheric components in the presence of sunlight to form photochemical smog, which is responsible for many respiratory problems.

Air Pollution and the Public

To the public, the most obvious effects of air pollution are the effect on visibility, and the effect it has on *them* – burning eyes, raw throats, and the like. The next most obvious effect is the "dirtying" of the public's environment – unpleasant air, smog, damaged paint and metal surfaces, etc. The damage that air pollution causes in plants is less visible. City dwellers may note the dirty leaves of the Norway maples in the park, but when they travel, the countryside looks clean and beautiful by comparison, and so many people are quite unaware of the increasing seriousness of air pollution damage to plants, particularly to crops.

Economic Importance and Assessment of Air Pollution Damage to Vegetation

A 1969 Stanford Research Institute study estimated that vegetation damage due to photochemical pollutants ran to nearly $113 million, including losses on ornamentals, non-commercial forests, parks, roadways, and residential plantings. While this is only 0.6 percent of the total crop value, it is a sizeable loss, and a loss that is growing annually. It should be noted, however, that these estimates were derived from 1969 census data. The estimate assumes that average climatic conditions prevailed during the growing season. Climate and growing conditions vary considerably from year to year and one would expect variations in crop losses accordingly. Current losses from air pollution damage to vegetation would amount to approximately $200 million based on more recent agricultural prices and production figures. Air pollution standards aimed at protecting vegetation are being adopted to arrest the trend, but here a problem arises: *Are the standards based on good data, and are they realistic?* The first order of business is to obtain good data, and that is what *assessment* is all about.

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*The earliest known air-pollution regulation was a royal proclamation by Queen Elizabeth I in the 16th century, prohibiting the burning of coal in London when Parliament was in session.

In an industrialized society, it is customary to base decisions of economic consequence on benefit/cost analyses. Dollar values often need to be placed on problems before resources can be allocated to solve them. The problem of pollution damage to plants has not escaped this custom. The "benefit" side of the argument says that pollution injury is so undesirable that it should not be permitted, regardless of the cost of control. The "cost" side of the argument says that pollution control equipment is very expensive, not only in terms of capital costs but also in terms of the additional human and management resources needed, and so enthusiasm for returning the environment to its pristine quality should be tempered by realism. But regardless of one's viewpoint, one thing is clear: to intelligently deal with the benefit/cost problems of pollution damage, we must first have a good way to *assess* the damage.

Air quality standards have been established to protect vegetation, but it is possible that some factors may have been overlooked or insufficiently studied, and that conditions may change over time, so that the standards may prove to be inadequate. Past surveys conducted in the various states have for the most part been limited in scope or breadth, and have been conducted with varying degrees of experience and knowledge, using a diversity of methods. A uniform system of data collection and interpretation would be useful in comparing the intrastate and interstate quality of the air, and more specifically, would be valuable in evaluating local losses that might or might not be associated with air pollution. We need a system for evaluating air pollution effects on vegetation that clearly determines the extent of damage, and identifies which pollutant is the culprit.

Air pollution survey techniques have ranged from the systematic to the guess. At one end of the scale, a systematic sampling of crops in affected fields is conducted, and the amount of leaf area injured on individual plants is calculated and used to determine reductions in yield. At the other extreme, damaged crops are simply inspected in the field, and a "guesstimate" is made of loss to the farmer. A number of states have conducted statewide surveys by simply enlisting the help of county agents or agricultural field inspectors to locate damage.

More recently, a systematic method of estimating and predicting losses was developed by the Stanford Research Institute. This model basically predicts the amount of injury on a county-by-county basis, based on the amount of fuel consumed, the presence of sensitive crops, and weather parameters. The model offers a simple approach to a complex problem, but it has not been field-tested extensively, and until additional ground surveys are conducted to determine whether refinements in the model are needed, its value will be limited. Comparisons have been made of the S. R. I. estimates and estimates from states that have conducted surveys, and although there is reasonable agreement in the estimates, a more refined model and better field assessment techniques are needed.

Direct Losses

For the most part, air pollution surveys have centered on direct losses. These are defined as tangible losses which are easily estimable. For example, a direct loss to a farmer would consist of damage that reduces the quality or quantity of his crops. Foliar injury to tobacco that downgrades the leaf from cigar-wrap grade to cut-tobacco grade is an example of reduction in quality. Leaf injury to potato vines resulting in smaller yields is an example of reduction in quantity. Direct losses may also take other forms – for example, processing costs may be increased if spinach or lettuce leaves are injured and have to be sorted to remove the unmarketable leaves before packaging.

Losses of this type are not restricted to food producers. Homeowners may experience losses with their garden crops or ornamental plants. A severely damaged white pine on someone's front lawn may have to be replaced because of its unsightliness. In this case, the direct loss would include the value of the tree, plus the labor involved in replacing it.

Indirect Losses

Indirect losses are less tangible, more difficult to assess, and have not received focus in the air pollution surveys. Indirect losses are those generated by or related to direct losses. For example, if a farmer can no longer grow a given crop on his land because of its sensitivity to ambient air pollution levels, he has little choice but to switch to another crop. The new crop may bring a lesser return to the farmer for his investment, or he may have to buy new or different machinery, or the new crop may not be as well adapted to the soil type. Although there is no pollution damage to the new crop, the farmer has still sustained a loss. On the other hand, the new crop may be better adapted and worth more, thus increasing a farmer's return on his investment.

Pollutants may predispose plants to insect or disease injury, forcing the farmer to spray or take other precautionary measures. Again, although the air pollutants have not in themselves caused direct, visible damage, the farmer has incurred a financial loss.

Perhaps the most serious indirect loss is that involving the reduction of property value. If farm land in an area becomes unproductive, or if production becomes economically unfavorable to the farmer, the land may be diverted to other uses. Indeed, it may be taken out of production and permanently committed to other uses. The general public is affected by such losses because the increased cost of production and processing are passed on to the consumer. Crops that can no longer be grown in one area may have to be transported from a more distant area.

Indirect losses may also affect the homeowner by decreasing the value of his property if ornamentals are injured or deformed by pollutants, or if his garden is in a known high-pollution area. Other indirect losses more difficult to assess include, among others, the deterioration of esthetically-pleasant landscapes, erosion following denudation of the land, stream silting, permanent injury to the soil by heavy metals, and deleterious changes in ecological systems.

Assessment Methods

To arrive at an accurate estimate of real economic loss, a number of variables must be considered in assessing crop damage. The time of the growing season during which injury occurs is particularly important. Plants injured early in the season may recover fully in a matter of days, provided the growing points have not been injured. If the plant is growing actively it may produce a new set of leaves and may recover in a few days, even after being completely defoliated by an acute fumigation. In this case, no real loss has been incurred, although the situation may have appeared disastrous immediately following the fumigation. However, if fumigation of this nature occurs later in the growing season, or if fumigations occur repeatedly, the plant may not be able to produce a new set of leaves and recover.

In evaluating air pollution effects on vegetation, it has been suggested that the term *damage* be reserved for cases where economic loss has occurred, and *injury* for cases where foliar or other effects are also manifested but no economic loss has resulted. Thus air pollution injury is defined as any identifiable and measurable response of a plant to air pollution. Damage is defined as any identifiable and measurable adverse effect upon the desired or intended use or desired product of the plant that results from air pollution injury.

Reduction in yield, whether it is quantitative or qualitative, is also of prime importance. Although estimation of yield loss may sound like a straightforward variable to measure, it can be very elusive. Foliar damage to root crops, for example, may bear no relationship to the amount of "economic" damage incurred. If injury occurs near harvest time, there may not be detectable yield loss. Sometimes, there may be a complete absence of foliar symptoms but a total loss of the crop – for example, if hay becomes contaminated with harmful quantities of fluorides, the crop may be worthless. It may be salvaged by diluting it with uncontaminated hay, or by using it as bedding. The important point to remember when confronted with such situations is that knowledge is required about *both* types of "damage" – the biological and the economic – and both should be reported.

The *end use* of the crop is also important, and the same injuries occurring to the same crop grown for different purposes may produce different economic results (and damages). For example, leaf scorch on a sugar maple in a forested area may not be of much economic consequence, whereas the same type of injury on sugar maples at a syrup-maker's could have a significant economic effect.

Whatever the economic factors involved, only three things need to be known in estimating crop losses: what crop is affected, to what degree, and by which pollutant. However, for these three items of information to be accurate, a great deal more information must usually be obtained and sifted. Sometimes repeated visits to an area will have to be made if the problem is chronic.

A model developed in California in 1970 uses a "rule of thumb" method. When field inspection of crops for air pollution damage revealed 1 to 5% leaf surface injury, then a 1% loss factor was applied for that crop in that particular county. When the injury ranged from 6 to 10%, a 2% loss factor was assessed. If the injury reached 11 to 15%, it became a 4% loss, and so on. The losses from air pollution for individual counties were then calculated from the harvest value of the crops by applying the appropriate percentage loss assigned to that county.

This procedure, and others like it, have been used by others. Although there is no theoretical basis for this technique, it does provide a systematic way of obtaining uniform data. But, further research is needed to establish the relationships among plant growth state, injury, and damage.

Field Inspection

There is no substitute for on-site field inspection to determine the extent of plant damage. For the field inspector to collect useful data, he must be able to *recognize* air pollution injury, and learn to properly *evaluate* the true impact of air pollutants upon plants. This book is designed to help do these two things. There are many difficulties that are encountered in the field which this book attempts to anticipate and offers guidelines to help the observer. In the discussion of so-called typical symptoms, for example, the term "typical" is used in the sense of a type specimen or as a composite about which variations occur. Therefore, typical should not be interpreted to mean "normal" or "usual" but used more as a point of reference in diagnosis.

Another important point to keep in mind is that there are many factors under field conditions that can modify or alter plant response to air pollutants. These can be categorized as follows:

- Biological character of plants
- Environment in which plants are growing
- Pollution concentration, and duration of exposure

The biological characteristics of plants include their genetic make-up and their stage of development. The sensitivity to various pollutants is known for many cultivated plants. Native vegetation and forest species may exhibit the gamut of response even among a given species, e.g., some pine trees will be injured while others growing a few yards away will be intact. This variation is simply a function of genetic variability which governs plant morphological, physiological, and biochemical characteristics. The stage of development of plants is important because some plants are not sensitive when young but become sensitive at maturity, and vice versa. Romaine lettuce is sensitive to PAN in the early stages of growth, becomes tolerant as the plant develops, and becomes sensitive again just before reaching maturity. Individual leaves also have sensitive and tolerant stages.

There are many environmental factors which affect plant response. These include climatic factors, light quality and quantity, temperature, relative humidity, and soil nutrition. In general, the conditions which tend to favor the growth of the plant also tend to increase their sensitivity to air pollutants. This aspect is covered in each chapter.

The concentration of the pollutant and the time during which the plant is exposed is referred to as the "dose," and the lowest dose that produces an effect is called the "threshold dose." However, because of the interrelationship of concentration and time, there is no single threshold dose for an effect to occur.

For example, plant injury may occur very quickly when exposed to a high concentration, but, at a low concentration, several hours may elapse before symptoms appear. This is not surprising since repair or detoxifying mechanisms are present in plants. There are some qualifications which should be made with respect to thresholds. Theoretically, exposure to a pollutant under controlled conditions will not produce injury on any individual plant until the dose exceeds a certain value – the threshold. Practically speaking, a plant is not exposed to increasing doses, but to one dose. Thus, for that individual, its threshold is unknown but is greater than the dose used if no injury occurs, or less than that dose if injury does occur. If several plants are exposed to the same dose under the same conditions, some may be injured and others may not. Thus the threshold or tolerance varies from individual to individual. If the dose is such that 50% of the individuals develop injury, it is called the *median effective dose* or *median toxic dose*. Moreover, if another group of plants is exposed to a dose that is half the median effective dose, 30%, 15%, or 3% of the individuals may be injured, depending upon the variability of tolerances for that group of plants. The frequency of symptoms on plants must be viewed as reflecting not only their average tolerance but also their heterogeneity.

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Chapter 2

STRUCTURE AND FUNCTION OF PLANTS IN RELATION TO AIR POLLUTION INJURY

Introduction

Higher plants, unlike most animals, are stationary organisms which utilize their surrounding environment for the synthesis of food and other life supporting processes. Because of their responsiveness to and dependence on their immediate environment, any shift in the usual environmental pattern tends to induce stress on the plant. For example, variations in temperature, water in the soil or air, minerals in the soil, toxic soil chemicals, diseases, insects, light characteristics, and gaseous air pollutants all have their influence. Response of a plant to stress depends upon a variety of plant characteristics such as age, metabolism, genetic makeup and structure. The purpose of this chapter is to consider certain aspects of plant structure, growth and physiology.

Higher plants usually have four basic organs: roots, stems, leaves and flowers. For the purposes of this chapter, discussion of each organ is undertaken separately and includes general morphology, function, internal structure, pattern of growth and, where appropriate, potential interaction with air pollutants. The common ultrastructure of cells of these organs is considered separately.

Plant Cell Ultrastructure

Chemical reactions in plants are not ubiquitous, but rather are localized, usually into some structural entity. The basic structural unit of plants is the cell. However, cells themselves are subdivided into many complex units, each associated with one or several functions. The following few paragraphs and associated figures discuss some of the basic structures of plant cells and the presumed function of these structures.

The outside boundary of each plant cell is the *primary cell wall* (Plate 2-1). This is the first formed wall of the cell and is deposited adjacent to pectic substances which separate cells and cement them together. This pectic layer is called the *middle lamella* (Plate 2-1). The primary cell wall consists mainly of carbohydrates and forms as the cell is initiated. Only in rare cases does the primary wall achieve any appreciable thickness. Some plant cells form a *secondary cell wall* (Plate 2-1) comprised of additional layers inside the primary wall. In this formation more carbohydrate strands are laid down. Lignin is then deposited between the strands of both the primary and secondary wall, forming a very hard layer. Lignification typically occurs after the cell has completed expansion and is responsible for the hardness of many plant parts.

A *plasmalemma* or plasma membrane occurs just inside the cell wall (Plate 2-2). This outer layer of the cell cytoplasm is chemically and structurally active, regulating the flow of ions, water and other material into and out of the cell. The plasmalemma and some other cytoplasmic components interconnect between adjacent cells through openings in the cell wall. The plasmalemma is the first living component of the cell exposed to air pollutants. Studies attempting to find primary sites of air pollution effects must consider the effect on the plasmalemma. Perchorowicz and Ting (1974) have found that ozone (O_3) alters the permeability of the membranes of bean leaf cells. The watersoaked appearance of leaves following exposure to certain pollutants is due in part to the disruption of the plasmalemma and other critical cell membranes.

The largest single component of the mature living plant cell is usually the *vacuole* (Plate 2-1), which occupies the center of the cell. Its main chemical constituent is water. Salts, organic acids, crystals and other metabolic products may also be present. The outer boundary of the vacuole is the *tonoplast* or vacuolar membrane (Plate 2-1). It functions much as the plasmalemma does in regulating the flow of substances passing between the cytoplasm and the vacuole.

The thin layer between the tonoplast and the plasmalemma comprises the *cytoplasm* (Plate 2-1), which contains many organized structures and is the site of most of the cell chemistry. The most prominent cytoplasmic structure is the *nucleus* (Plate 2-2) which houses most of the genetic system of the plant. The denser structures seen in the nucleus in Plate 2-2 are *chromatin* which make up the *chromosomes.* The genes which regulate specific traits of the plant are found here. Surrounding the nucleus and regulating movement of chemicals in and out is the *nuclear envelope* (Plate 2-2). Evidence that air pollutants or their secondary products can penetrate the nuclear envelope and alter the genes or chromosomes, thus causing inheritable genetic change, is not available at this time.

Several other organelles occur within the cytoplasm. Of these, one group unique to plant systems is the plastid. The most familiar plastid is the *chloroplast* (Plate 2-3). The chloroplast is surrounded by a double unit membrane. The internal membrane invaginates, forming layers of lamella (Plate 2-3) or *thylakoids.* At intervals, stacks of thylakoids (10 to 100) form cylinder-like structures called *grana* where most of the photosynthetic reactions take place. A matrix, the *stroma,* surrounds these membranous structures.

During the process of photosynthesis light energy is absorbed by chlorophyll and other pigments, and used to form sugars from carbon dioxide (CO_2) and water (H_2O). This process is the only biologically significant way of converting CO_2 to usable organic matter for living organisms. Generally speaking all of the oxygen (O_2) occurring in the earth's atmosphere is released from the breakdown of H_2O during photosynthesis.

All of the major air pollutants have been shown at some dose level, to slow or stop photosynthesis. The rate of photosynthesis is in part dependent upon adequate gas exchange between cells containing chloroplasts and the outside atmosphere. Therefore, any plant response to air pollution which restricts gas exchange will inevitably slow the rate of photosynthesis. This problem will be considered more fully during a discussion of gas exchange in leaves. The amount of chlorophyll may also regulate the rate of photosynthesis. Chlorosis in leaves indicates that less chlorophyll is present. Chlorophyll is unstable and normally is continuously replaced within the chloroplast. Chlorosis may result from a direct attack on the chlorophyll molecule, as it seems to be with sulfur dioxide (SO_2)

and possibly fluorine (F), or it may result from inhibition of the chemical pathway which produces chlorophyll, as it seems to be with F, O_3 and peroxyacetyl nitrate (PAN). Photosynthesis is also slowed when the chemical energy used for CO_2 fixation is in some way tied up. Any form of pollutant which can enter the chloroplast and serve as an electron acceptor is potentially capable of this type of reaction. Rapid resumption of the rate of photosynthesis following an air pollution exposure indicates interference by the pollutant with the energy exchange mechanism in photosynthesis or with regulation of the opening of stomata, thus inhibiting gas exchange. Longer recovery times are required when critical molecules and structures must be resynthesized.

During photosynthesis light energy is converted into chemical bonds of organic molecules. Utilization of the chemical energy in these bonds requires oxidation. Respiration is the term applied to these oxidation processes. The initial respiratory steps occur in the cytoplasm. Products of the initial steps then pass into *mitochondria* (Plate 2-2) where they are further oxidized forming molecules of the important energy carrying compound adenosine triphosphate (ATP), a variety of organic acids and CO_2. Oxygen serves as the final electron acceptor and H_2O is also formed within the mitochondrion. As in photosynthesis, gas exchange both within the cell and with the outside atmosphere is necessary in respiration. Air pollutants which inhibit gas exchange may inhibit respiration. Ozone at low levels may cause mitochondria to increase their rate of O_2 uptake after an initial inhibition (MacDowall, 1965). Other pollutants also affect the rate of respiration.

The plant cell utilizes the products of photosynthesis and respiration to construct all of its components. Proteins, most important as enzymes and components of membranes, are formed within the cytoplasm. This synthesis is subject to interference by some air pollutants. Lipids, most important as components of membranes, are also synthesized in the cytoplasm. Air pollutants are known to attack the lipid portion of membranes, so lipid synthesis becomes very important in repairing injury to membranes caused by air pollution. Complex carbohydrates and other cell chemicals are also formed in the cytoplasm, usually in association with some specific cell organelle. Since, as yet, air pollution has not been shown to interfere directly in these processes, their associated structures will not be considered. However, cell systems are very interdependent. Effects on one component are often reflected in other components.

Plant Organs

The following discussion covers the structural organization of the four basic plant organs and considers potential interactions of these structures with air pollutants.

Roots

Roots function in the absorption of water and nutrients from the soil. They often store foods, transport water and nutrients to the shoot, and anchor the plant in the soil.

To accomplish these functions a variety of root forms exist. These forms are usually categorized into four groups. One of the common root types is the *tap root.* The tap root is typically a single large root extending directly down from the plant axis. It may penetrate deeply into the soil providing an avenue for absorption of water from depths of twenty feet or more, such as it does in alfalfa or some desert plants, or it may be more fleshy in form providing extensive storage space such as in carrot.

A second type of root system is the *fibrous root* system. In this system, roots branch frequently and tend to remain quite small in diameter. Very dense mats of roots are often formed. Many grasses have fibrous root systems as do shrubs such as rhododendron.

Fascicled roots constitute an intermediate form between the tap root and the fibrous root system. Here a large, fleshy, but branching system is produced. An example is the sweet potato.

Finally, the fourth root system type is one which is produced by stem or leaf tissue. This type, the *adventitious root* system, is often fibrous, but may be quite variable. In corn the original root system dies and adventitious roots are formed from the lower nodes of the stem. Such roots are sometimes called *prop roots.* Many grasses produce roots from their underground stems. Some ivys produce adventitious roots which aid in clinging to other structures. Orchids have adventitious roots which absorb water and nutrients from the air. The ability of plants to produce adventitious roots is utilized in vegetatively reproducing plants via cuttings or air layering.

Root Tissues

Specific functions within roots are generally relegated to specific tissues. Absorption initially occurs in the outer layer of the root called the *epidermis* (Plate 2-4) in young roots. Usually many of these epidermal cells are extended, forming *root hairs.* Such a system increases the surface area of the root, thus providing a larger absorptive area.

Typically the tissue just inside the epidermis is the *cortex* (Plate 2-4). Cortical tissues often store food, water, or both, provide wall surfaces for water and nutrient transport, and intercellular spaces for gas exchange across the root and into the shoot.

Separating the stele from the cortex is the *endodermis* (Plate 2-5). This single layer of cells forms a cylinder which serves to regulate the passage of materials into and out of the *stele* (all tissue inside the endodermis).

Inside the endodermis is the *pericycle* (Plate 2-5), the site of initiation of lateral roots and in some instances part of the vascular cambium. During lateral root initiation a new root apical meristem forms and grows outward through the cortex (Plate 2-7).

The internal tissue of the root is most commonly *xylem* (Plate 2-5), although some roots do contain a pith. Xylem is the water and nutrient conducting tissue of the plant where the direction of transport is typically upward. *Phloem* (Plate 2-5), also occurs in the stele. Phloem is the manufactured food conducting system and the direction of flow is usually downward toward the roots. In general one would expect water and minerals to move out of the soil, through the epidermis, cortex, endodermis, pericycle and finally into the xylem where they would be transported to the rest of the plant. Sugars and other manufactured products move out of the leaves in phloem cells and are transported throughout the plant, including the roots, where they move through the pericycle and endodermis into the cortical cells for storage or use.

Root Growth

Growth in roots occurs in two major ways. *Primary growth* results from cell production at the tip of each root and the subsequent elongation of these cells. This elongation occurs near the root tip and precedes the formation of root hairs and lateral roots. The region which forms the new cells is called the *root apical meristem* (Plate 2-6). An additional tissue layer is produced by the root apical meristem, the *root cap,* which surrounds and protects the root apical meristem as the root tip grows through the soil.

Some roots exhibit an additional pattern of growth called *secondary growth.* In this case a cylinder of dividing cells, the *vascular cambium* (Plate 2-7), develops between the xylem and phloem and produces more vascular tissue (xylem and phloem). The vascular cambium may remain active for as long as the plant lives, producing new layers of xylem and phloem each year, which connect with the new vascular tissue produced at the root and shoot tip. The activity of this cambium, like that of the apical meristem, is dependent upon energy and building materials from the shoot. Any restrictions placed on the metabolism (e.g. photosynthesis) of the shoot, such as those which might result from air pollution, can be expected to reduce the activity of these two meristems resulting in less root growth.

Since the dermal and cortical tissues cannot expand significantly, plants which produce extensive secondary tissue also produce a *cork cambium* (Plate 2-7) outside the stelar tissues. This layer is part of the bark and functions primarily in protection.

Certain plants called *legumes* may become infected with microorganisms such as bacteria. The infection occurs through a root hair in the case of bacteria, and results in the formation of a growth on the root called a *nodule.* The bacteria within the nodule have the ability to change atmospheric nitrogen into a form usable by plants. This is an important source of necessary nitrogen for crop plants and for plants in natural ecosystems. In studies on the effect of O_3 on legumes it was found that nodule numbers were decreased by exposure to this pollutant, but their ability to fix nitrogen was not curtailed. The net result, however, was a decrease in total nitrogen fixed. It was presumed that the effect resulted from a deficiency of food coming from the shoot due to inhibition of photosynthesis and was not due to any direct effect of O_3 on the roots or bacteria. Similar responses can be expected from plants which associate with nitrogen fixing blue-green algae.

Another instance in which a lack of adequate photosynthate may reduce root growth was presented by Tingey, *et al.* (1971) where marked effects occurred on root and hypocotyl growth without visual injury to the shoot. Over the growing season the effect of a reduction in root mass was a reduction in shoot productivity, since presumably less water and nutrient came from the soil into the plant.

Stems

Stems are the supporters and producers of leaves and flowers. They provide a transport system for water, minerals and manufactured food products between the roots and other plant organs. To a certain extent stems may be storage organs and in many herbaceous plants they are photosynthetic. In general morphology, stems are composed of *nodes,* sites where leaves are attached, and *internodes,* areas of the stem between nodes. They usually possess a *terminal bud* at the tip and *axillary buds* in the axils of each leaf. The axillary buds may produce stems or flowers.

Stem Tissues

The form and tissue organization of stems is extremely variable. However, there are several common types. One type is the *herbaceous dicot* stem (Plate 2-8), such as is found in alfalfa or squash. These stems are small in circumference and rather succulent. In cross section the xylem and phloem are localized in discrete *vascular bundles* (Plate 2-8) in a concentric ring near the stem periphery. As in the root, a *cortex* and *epidermis* surround the vascular tissue, but the cortex is much less extensive. The epidermis often has stomata, trichomes and a waxy cuticle. Much of the stem is occupied by a central pith area which serves in storage. The main distinguishing features of the herbaceous stem are its small size and small amount of vascular tissue as compared to *ground tissue* (pith and cortex).

A second stem type is the *woody dicot* (Plate 2-9). In its initial development it too has vascular bundles in a concentric ring. However, secondary growth is initiated very early in the stem's ontogeny, producing extensive xylem and phloem. The new vascular tissue forms a continuous cylinder which is the characteristic growth ring of woody plants. The term *wood* applies to xylem tissue which surrounds a small amount of *pith. Bark* includes the phloem, cortex and epidermis. The epidermis and cortex are similar to that found in herbaceous dicots. As secondary growth proceeds a *cork cambium* is initiated and gives rise to the corky cells characteristic of the outer layers of bark. In certain areas of the stem bark, clusters of loosely associated cells form a structure called a *lenticel* (Plate 2-18). This structure provides a means of gas exchange for the inner living stem tissues. Bands of living cells extend laterally within the wood and bark. These bands, the *rays,* are responsible for moving material across the wood and bark, and for storage.

Gymnosperms such as *pine* (Plates 2-10, 2-11), a third stem type, have a similar tissue arrangement as the woody dicot. The main distinctions are differences in cell types in the vascular tissue and the presence of *resin ducts.*

A final stem type, the *monocot* (Plate 2-12), is exemplified by plants such as corn, wheat, oats and other grasses. These plants are usually herbaceous and relatively small. The stems are often telescoped, at least until the time of flower production. Often, lateral stems are produced. If they grow laterally underground they are called *rhizomes,* while those stems growing above ground laterally are called *runners* or *stolons* (these terms apply to all plants producing this type of branching). The internal structure of monocot stems is composed of *vascular bundles* irregularly arranged in parenchymous *ground tissue.* An epidermis with stomata occurs around the outside and often is accompanied by one or several layers of cells which may be lignified. Frequently the vascular bundles turn laterally and intermix at the nodes, producing a very disorganized configuration. Many monocot stems are green, with the chlorophyll-containing cells in the ground tissue near the periphery of the stem.

Stem Growth

Growth in stems occurs as it does in roots at several sites. *Primary growth* results in an increase in stem length and occurs as cell division and elongation at the stem tip. This growing region is called an *apical meristem* (Plate 2-13). It differs from the root apical meristem in that it does not possess a root cap and it produces leaves and lateral buds which become the apical meristems of vegetative or floral branches. Many stems further increase their length by retaining meristematic tissue (*intercalary meristems,* Plate 2-13) just above each node. An example of such growth is in smooth cord grass, in which all the leaves are present when the entire stem is a few centimeters (1 to 2 inches) long. After the terminal inflorescence is produced the intercalary meristems become active, forming the long internodes characteristic of this grass plant.

Secondary growth is an additional facet of plant expansion for many stems. A vascular cambium (Plate 2-11) may form between the xylem and phloem. This layer forms more vascular tissue and thereby causes lateral expansion of the stem. Accompanying the production of more vascular tissue is the activation of a *cork cambium* (Plate 2-11), usually in the outer ground tissue. Divisions in this tissue result in the maintenance of a protective corky layer and some storage tissue outside the phloem of the bark. As previously implied, these structures may remain active for many years in woody perennials, producing new tissues which are continuous with the new primary growth. Many annuals also possess cambia which increase the vascular tissue as the plant expands during the growing season. Most monocots do not have secondary growth.

It is apparent that to increase the mass of shoot tissue in a characteristic way the apical meristem must function normally. This function requires a critical balance of food for energy, nutrients, water and growth regulators. Air pollutants may upset the critical balance between these components, thus producing a plant abnormal in size or shape. Several air pollution studies have attempted to use the yearly increment of wood as an indicator of stress caused by air pollution. While such studies do indicate varying degrees of growth from year to year, it must be noted that variation occurs naturally due to differences in moisture and other environmental parameters. Although the accuracy of this method has been questioned, it is nevertheless often possible to demonstrate significant growth reduction effects associated with some air pollution sources, when compared to proper control areas or pre-pollution growth rates at the same sites.

Leaves

The primary function of leaves on most plants is photosynthesis. Leaves are uniquely designed to accomplish what may be the most important of all biological processes. In addition, some leaves store food, water, or both. A few leaves are involved in asexual reproduction, and some in protection.

There are a few general terms for various leaf parts and leaf arrangements. The leaves of many broad-leaved plants are composed of a *petiole,* the stemlike portion, and a *blade* or *lamina,* the flattened portion. The leaves of plants such as grasses also have a blade, but that portion corresponding to the petiole is flattened and often encircles the stem. This flattened portion is called a *leaf sheath.* The arrangement of leaves on a stem may be *alternate,* one leaf per node, *opposite,* two leaves per node, or *whorled,* with more than two leaves per node. A leaf may be simple with a single blade per petiole, or compound with several leaflets per petiole. This determination is not always easy, but the distinctive feature is the presence of a lateral or axillary bud at the base of the petiole. The individual leaflets of a compound leaf do not have an axillary bud. There is one axillary bud for the entire compound leaf located at the base of the petiole, just as it is in simple leaves. In many plants, such as soybean, the first foliar leaf is simple with later leaves being trifoliate (compound). Leaves of the common gymnosperms are frequently needle-like. They may be born singly or in groups in a *fascicle.* The base of each fascicle has an enclosing *sheath.*

Leaf Function

The function of leaves has no doubt accounted for the many structural variations which occur in nature under different environmental conditions. The primary function of most leaves is photosynthesis. As has already been stated, this involves the incorporation of atmospheric CO_2 into carbohydrates within the chloroplasts and the concomitant release of O_2 back into the atmosphere. The gas exchange occurs as the result of diffusion through openings in the leaf called *stomata* (Plate 2-17). When photosynthesis is occurring, CO_2 enters the water solution in the leaf mesophyll cell walls and diffuses, probably as carbonic acid, into the chloroplasts. This uptake of CO_2 from the intercellular spaces of the leaf lowers the CO_2 concentration and provides the diffusion gradient necessary for a net flow of CO_2 into the leaf. At the same time O_2 leaves the chloroplasts because of the high concentration there, enters the intercellular spaces and finally diffuses into the outside atmosphere where the concentration is lower. This pattern of gas exchange would typically occur during daylight hours when photosynthesis is occurring. At night one would not expect a net CO_2 uptake nor O_2 liberation with most plants.

One problem which arises from this mechanism of gas exchange involves water. Each cell wall of the leaf mesophyll is saturated with water and at least partially exposed to intercellular spaces. This means that water will evaporate from the cell wall surfaces and produce a saturated atmosphere within the leaf. The relative humidity (RH) of the intercellular spaces of a leaf is usually higher than that of the outside atmosphere if the temperatures are equal. Therefore, if the stomata are open, water vapor diffuses from the leaf at a more rapid rate than it diffuses in. This process is called *transpiration*. Temperature influences the rate of molecular movement and thus the rate of diffusion. True transpiration rates are calculated on the basis of vapor pressure gradients, which account for both RH and temperature differences between the leaf and the outside atmosphere. Water vapor lost by the plant is replaced with water from the soil. As a water molecule evaporates from the cell wall of a leaf it pulls another water molecule behind it, thus providing the energy to transport water. Minerals and other material entering the roots are also moved up the plant in xylem cells in this stream of water.

Since water loss is such a general problem for plants, it is not surprising that a variety of mechanisms have evolved to conserve water. For instance most plants close their stomata at night. This reduces water loss and also prevents the substantial uptake of most air pollutants. The act of wilting closes stomata as a general rule. Plants in a wilted condition are not very susceptible to air pollution. Some plants have stomata sunken into the mesophyll tissue which tends to reduce the rate of gas exchange and therefore pollutant uptake. Many desert plants have very tightly packed mesophyll cells, reducing the surface area for evaporation. These plants are generally quite tolerant of air pollution. The deciduous nature of many plants can be considered water conserving. Evergreens such as pines, though adapted to conserve water, may be more susceptible because their leaves may receive pollution the year round.

Many air pollutants enter plants through leaf stomata. The degree to which the stomata are open is dependent upon how much water is in the guard cells surrounding the stomatal aperture. As the guard cells fill with water, the aperture gradually increases in size. The reasons for water influx are currently thought to be related to the production of organic acids from insoluble starch in the guard cell and the concurrent movement of potassium ions (K^+) into the guard cells. The accumulation of these ions effectively lowers the water potential of the guard cells and causes water to move in. The chemistry of this phenomenon is not absolutely clear, but certain facts are known. The presence of light stimulates organic acid formation and K^+ movement into guard cells resulting in stomatal opening. Darkness reverses the process. The concentration of CO_2 outside the leaf and in the internal tissues of the leaf can regulate cell pH and thus organic acid formation and K^+ flux. Therefore, low leaf CO_2 content promotes stomatal opening. Low temperature (0°C) can override the light mediated stimulation and thus cause stomata to remain closed. Very high temperatures (40°C) also inhibit stomatal opening in light. Intermediate temperatures may induce a range of degrees in the size of the stomatal aperture. The closing of stomata during midday periods may be the result of this temperature phenomenon. Lack of water in the leaf induces stomatal closing in light. At times when transpiration exceeds water uptake, leaves wilt and stomata close.

The uptake of any air pollutant into a leaf is dependent not only on stomatal opening but on what happens to the pollutant once it reaches the cell surface. If the pollutant moves into the cell quickly, the diffusion gradient will be favorable for more pollutant uptake into the leaf. The converse is also true. The rate of movement of a gas into and out of a leaf is dependent upon stomatal opening, temperature, and diffusion gradient and is independent of the rate of movement of any other gas.

Leaf Structure

The outer covering of the leaves of *broad-leaved* plants is usually a single cell layered *epidermis* (Plate 2-14). These epidermal cells are covered by a waxy *cuticle*, which varies in thickness according to species and environment. Hairs or *trichomes* are common, protruding from the leaf surface. Stomata also occur in the adaxial (upper), abaxial (lower) or both epidermal layers.

Beneath the adaxial epidermis is one or more cell layers in which the cells are perpendicular to the epidermis. These cells of the *palisade layer* (Plate 2-14) often appear tightly packed when viewed in cross section, but in reality have air spaces surrounding each cell. Beneath the palisade layer, cells become irregular in their orientation. This mass of tissue comprises the *spongy layer* (Plate 2-14). Together the palisade and spongy layers make up the leaf *mesophyll*, which is the tissue of the leaf involved in photosynthesis.

Embedded in the leaf mesophyll is the *vascular bundle* or vein system. The branching arrangement of the veins is variable, depending on the species. The major veins contain xylem and phloem and a certain amount of tissue which supports the leaf. As branching continues there is progressively less tissue until only a few xylem cells are left in the smallest veins. In some plants the vein endings occur at the margins of the leaf, while in others they are scattered throughout the leaf, a factor which can influence the location of pollution injury.

When veins end at the margins or tips of leaves there is often a cluster of loosely associated cells, the *epithum*, just beyond the last xylem cell. The epidermis at this point has a stomata-like structure in which the guard cells do not function, leaving the aperture open at all times. This entire structure is called a *hydathode*. When stomata are closed, the water taken into the plant can exceed that which is released. This tends to build up internal pressure in the xylem called *root pressure*. Under these circumstances water droplets are exuded from the leaf hydathodes, in a process called *guttation*. Droplets seen on leaves during the night and early morning are often guttation droplets rather than dew. These droplets make excellent collectors of air pollutants. At times toxic solutions can build up in these droplets and can cause severe lesions on the leaf margin. Plants like blackberry and strawberry are good indicators of this type of response in the field.

A second leaf type characteristic of many grasses is that of corn, sometimes called a *narrow-leaved* plant (Plate 2-15). This leaf too has a single epidermal layer with a covering cuticle. Stomata are present in both leaf surfaces although in many leaves of this type only the upper epidermis has a significant number. Trichomes are also common.

The *mesophyll* (Plate 2-15) of corn and other grasses is not divided into a palisade and spongy layer, but is a mass of rather irregularly arranged cells. In some narrow-leaved plants the mesophyll cells are oriented perpendicular to and in a ring around the veins.

The vascular bundle in corn is surrounded by a *bundle sheath* which encloses the xylem and phloem. As in the broad-leaved plants there are major veins with extensive branching. However, the major veins of corn and other grasses run parallel to each other from the base to the tip of the leaf. Air pollution injury is common in such leaves at the tip or as lesions running parallel to the veins.

The *needle-like* leaves of gymnosperms such as pine have a somewhat more complex structure (Plate 2-16). The epidermis is covered with a thick cuticle. In some instances the outer walls of the epidermal cells are lignified. Stomata occur in rows along the length of the leaf and are sunken into the mesophyll tissue. Usually hairs and wax occur in stomatal depressions.

Beneath the epidermis occurs a layer of small lignified cells, the *hypodermis*, and subtending these are several layers of *mesophyll* cells. These mesophyll cells are rather compact with limited intercellular spaces. The mesophyll terminates at the *endodermis* which is composed of a single layer of cells. Inside the endodermis are several layers of larger, thin walled storage cells comprising the *transfusion* layer. Embedded in the center is a vascular bundle with one to two areas of xylem and phloem. Large *resin ducts* are common in the mesophyll with smaller ones occurring inside the endodermis.

Stewart, *et al.* (1973) attempted to use tissue analysis in conifers to distinguish the cause of injury. They had some success in narrowing the cause down to two or three possible agents if the stress was not too severe. More recently Evans and Miller (1975) report that they are able to determine whether injury to ponderosa pine leaves is caused by O_3, SO_2 or winter injury by determining which cells of the leaf are injured and how the cell organelles respond.

Leaf Growth

Some of the metabolic characteristics influencing variability in injury responses to air pollution in leaves are due to age differences of individual cells. Many of the cell divisions involved in leaf formation occur before the leaf can be seen. The major expansion of many leaves is due to cell enlargement, plus the formation of intercellular spaces.

Broad-leaved leaves most commonly grow first with apical growth. The flat nature of the leaf develops when activity begins to occur in lines marking the presumptive leaf margins. The actual shape is regulated by *marginal* and *submarginal meristems.* As growth continues and the shape is determined, many of the young leaf cells divide to produce the final mass of the leaf. Interference with these divisions or those of the marginal and submarginal meristems produces the savoying and cupping caused by some air pollutants.

Leaves of corn or other grasses have early apical growth, but subsequently most of the new growth is basal via two *intercalary* meristems. One of these meristems is located at the base of the blade while the other is located at the base of the sheath.

Pine needles also elongate initially by apical growth, with intercalary meristem activity at the leaf base producing most of the actual needle. Bands of injury caused by air pollution are more likely to develop in leaves of pine or narrow-leaved plants where gradients of tissue age occur along the length of the leaf.

Flowers, Fruits and Seeds

A discussion of floral structure is beyond the scope of this text, but several features of this organ and its function relate to air pollution injury. First the longevity of the flower itself is rather short as compared to other organs. This means that the time available for direct acute pollution injury is less than for other organs. This is not to say that effects are less likely, since the fruit and seed are directly dependent on the rest of the plant for their growth and development.

In many plants the stimulation to produce a flower comes from the leaves. The leaves may measure the length of day through the reaction of the pigment *phytochrome* to light and darkness. Once a certain biochemical state is achieved the shoot vegetative meristem is converted to a floral meristem and flower production begins. It is possible for stress such as air pollution to upset the timing of this process.

Once flower development is complete pollination can occur. The male sex cell is carried in pollen grains by wind or insects in many plants. Pollen seems to be very tolerant of air pollution. Germinating pollen grains may be injured when exposed to pollution in dishes in a chamber. Reduction of the yield of corn in the field has been attributed in one instance to the interference of air pollution in the successful growth of pollen grains. If changes occur in the chromosomes of sperms in pollen grains, these changes may show up as different traits in the next generation. A more likely result would be sterility with no embryo formation.

Fertilization takes place deep within the floral tissue and is probably well protected from direct air pollution injury. However, the growth of the embryo, the seed and the fruit may be subject to effects indirectly produced by air pollution. These structures are dependent upon the rest of the plant for the food which builds their mass. If, at the time when these tissues are building, leaf tissue is severely injured, the metabolites which normally go to the flower may not be available. Heagle, *et al.* (1974) have shown that reduction in soybean yield can occur if O_3 is administered at a time when the flower is developing the fruit and seed. Exposures early in the life cycle allowed time for the plant to recover, while later exposures had no effect on yield. Taylor, *et al.* (1958) have shown a reduction in avocado fruit yield due to photochemical oxidant in southern California. In this case there were no visible symptoms, but merely a reduction in photosynthate available to form fruit mass.

The petals of flowers often have thin cuticles and may be sensitive to air pollution. In the cut flower industry pollutants like F are damaging to plants such as gladiolus. Ethylene also is potentially destructive to flowers such as orchids.

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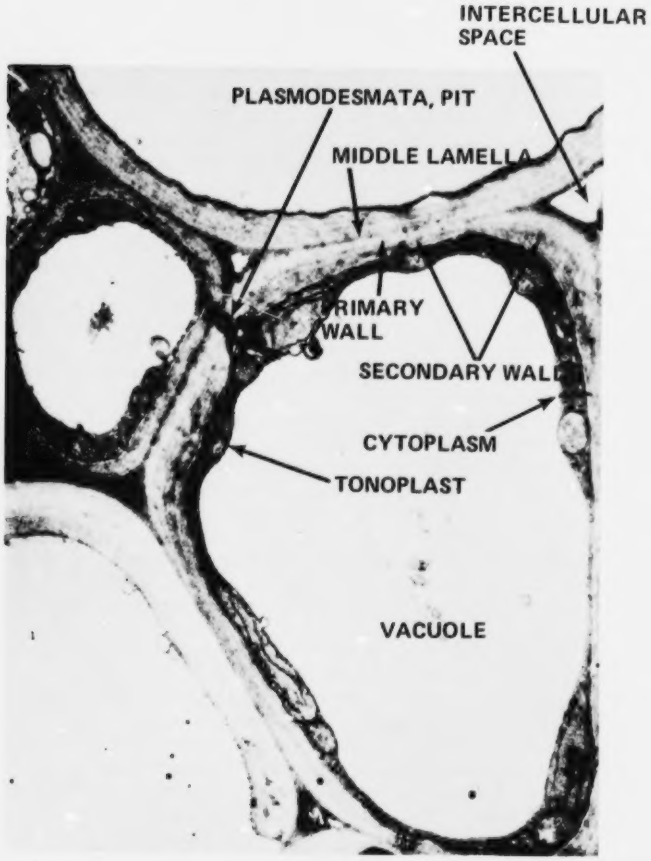
INTERCELLULAR SPACE
PLASMODESMATA, PIT
MIDDLE LAMELLA
PRIMARY WALL
SECONDARY WALL
CYTOPLASM
TONOPLAST
VACUOLE

Plate 2-1. *Leaf Cells of Spartina alterniflora (Smooth Cord Grass) Showing Cell Organelles*

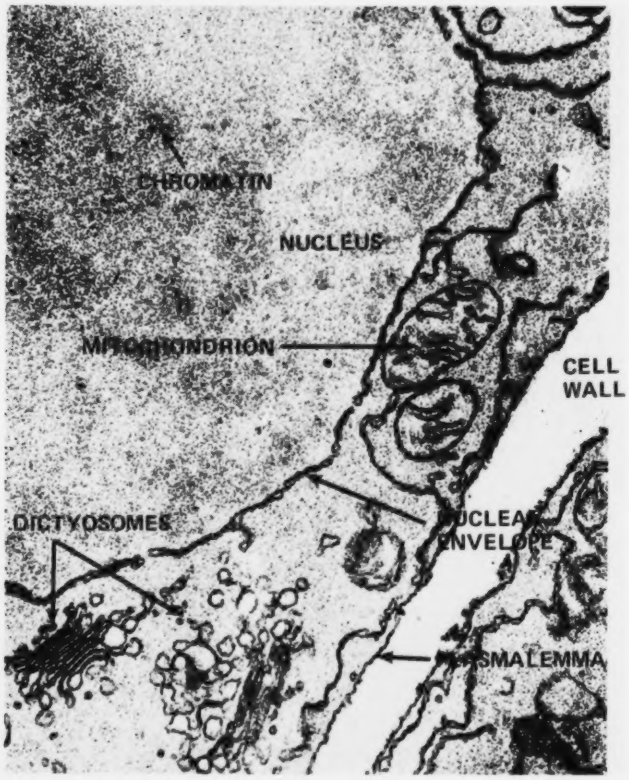
CHROMATIN
NUCLEUS
MITOCHONDRION
CELL WALL
DICTYOSOMES
NUCLEAR ENVELOPE
PLASMALEMMA

Plate 2-2. *Portion of Root Meristem Cell of Chrysanthemum Showing Cell Organelles*

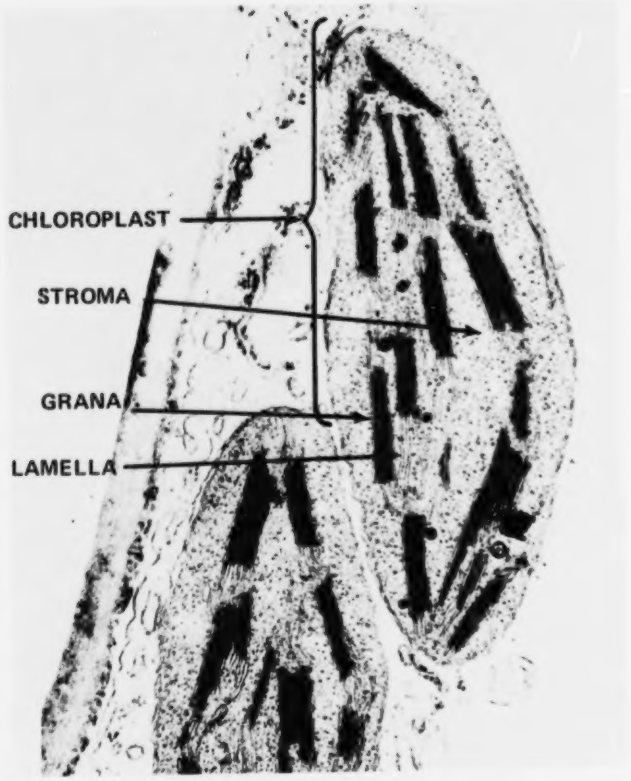
CHLOROPLAST
STROMA
GRANA
LAMELLA

Plate 2-3. *Section Through Chloroplasts of a Chrysanthemum Leaf Cell Showing Details of Grana and Lamella*

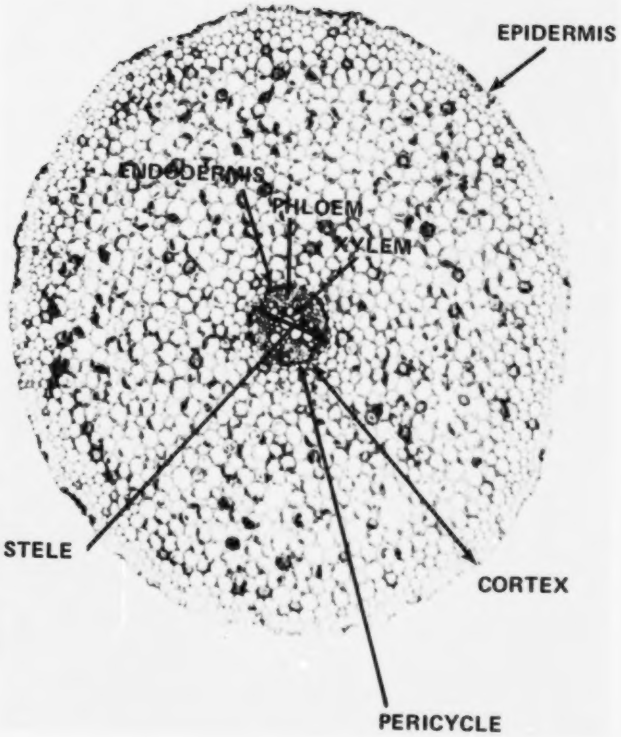
EPIDERMIS
ENDODERMIS
PHLOEM
XYLEM
STELE
CORTEX
PERICYCLE

Plate 2-4. *Cross Section of a Ranunculus sp. (Buttercup) Root*

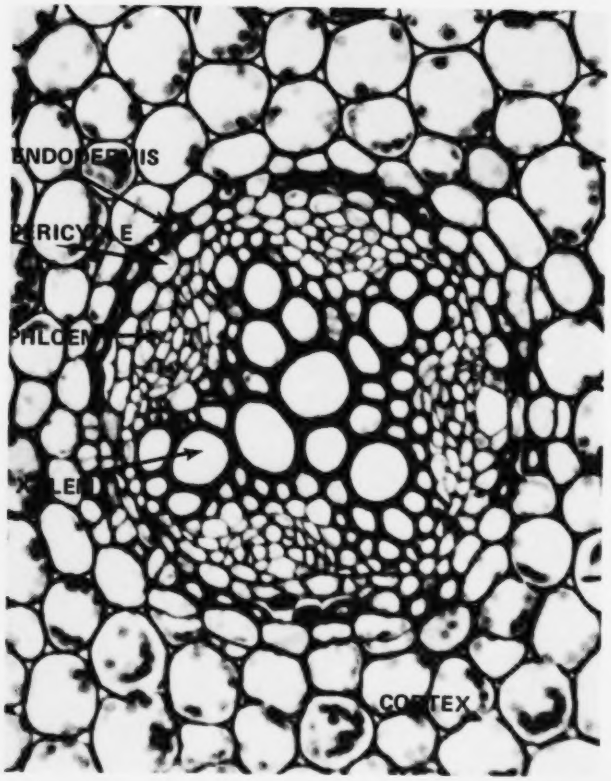
ENDODERMIS
PERICYCLE
PHLOEM
XYLEM
CORTEX

Plate 2-5. *Cross Section Showing the Stele Area of a Ranunculus sp. (Buttercup) Root*

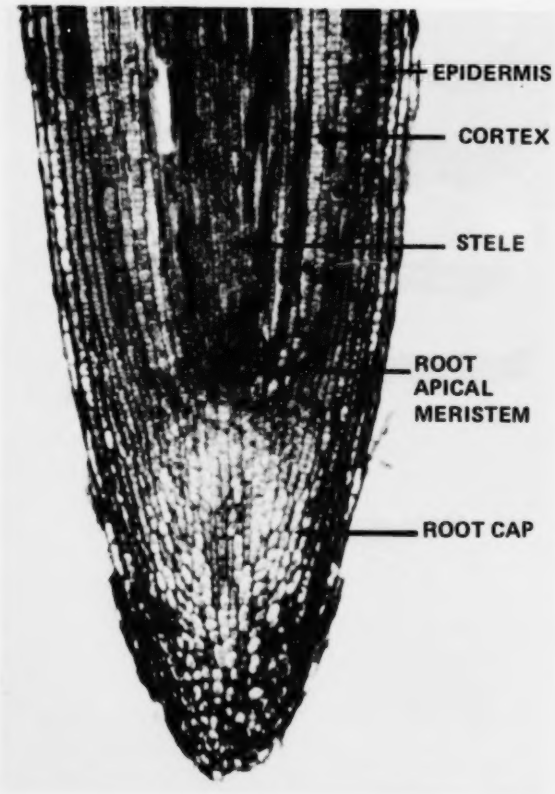
EPIDERMIS
CORTEX
STELE
ROOT APICAL MERISTEM
ROOT CAP

Plate 2-6. *Longitudinal Section Through the Apical Meristem of Strophostyles helvola (Beach Pea) Root Showing Area Where Primary Growth Occurs*

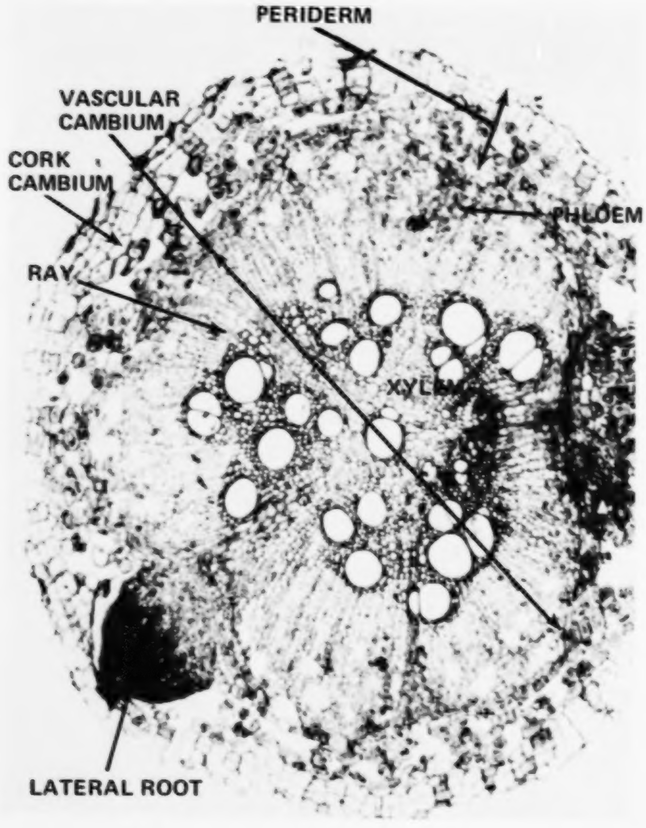
PERIDERM
VASCULAR CAMBIUM
CORK CAMBIUM
PHLOEM
RAY
XYLEM
LATERAL ROOT

Plate 2-7. *Cross Section of the Root of Nicotiana tobaccum (Tobacco) Showing Secondary growth and Lateral Root Formation*

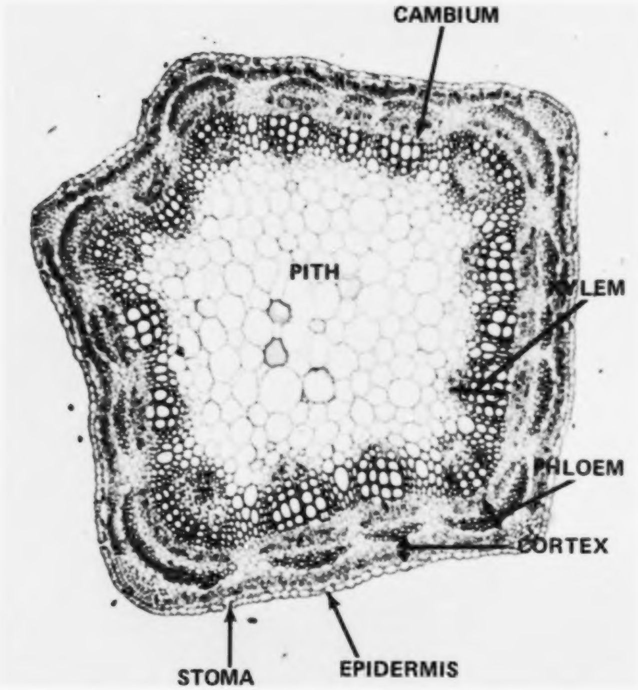
CAMBIUM
PITH
XYLEM
PHLOEM
CORTEX
STOMA
EPIDERMIS

Plate 2-8. *Cross Section of the Herbaceous Dicot Stem of Medicago Sativa (Alfalfa)*

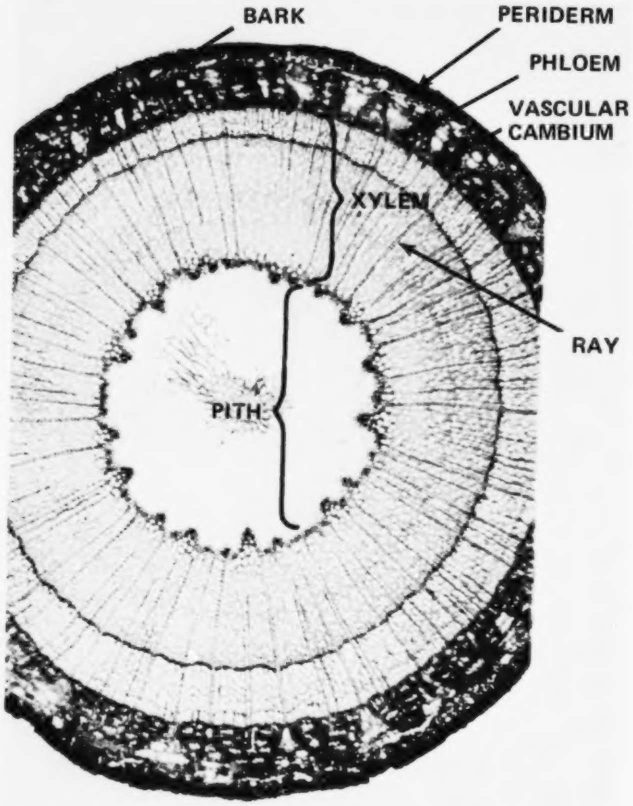
BARK
PERIDERM
PHLOEM
VASCULAR CAMBIUM
XYLEM
RAY
PITH

Plate 2-9. *Cross Section of the Woody Dicot Stem of Liriodendron tulipifera (Tulip Poplar)*

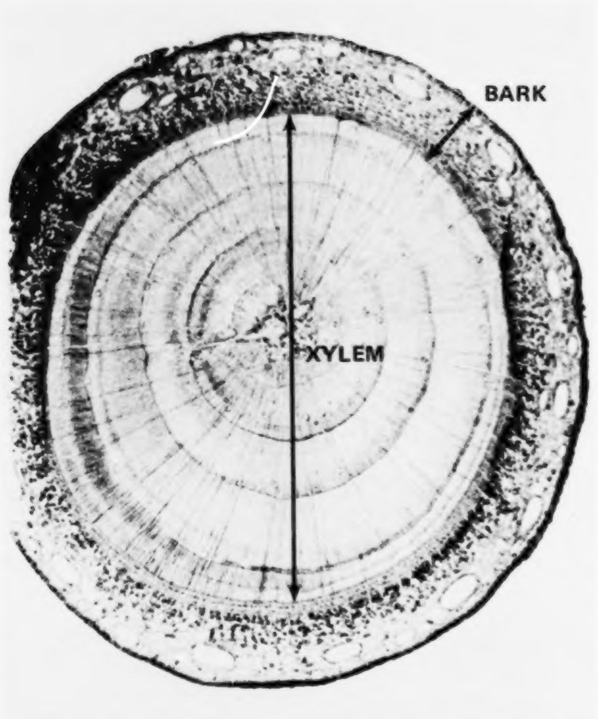

Plate 2-10. *Cross Section of the Gymnosperm Stem of Pinus sp. (Pine)*

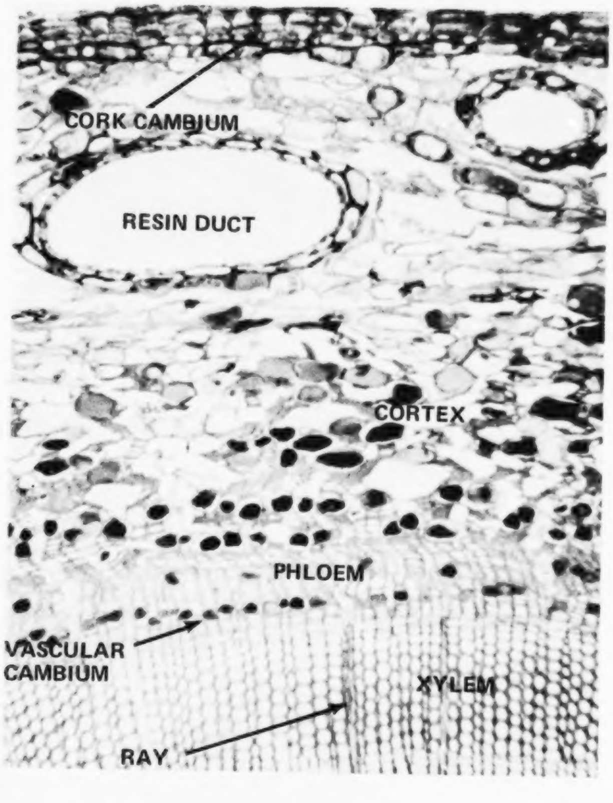
CORK CAMBIUM
RESIN DUCT
CORTEX
PHLOEM
VASCULAR CAMBIUM
XYLEM
RAY

Plate 2-11. *Cross Section of the outer area of the Gymnosperm Stem of Pinus sp. (Pine) Showing Sites of Secondary Growth*

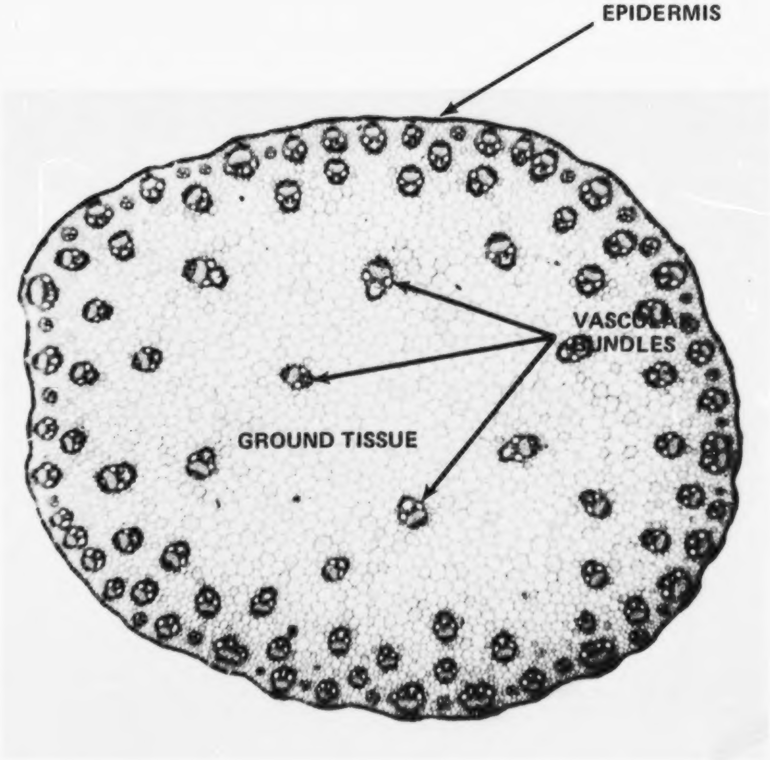
EPIDERMIS
VASCULAR BUNDLES
GROUND TISSUE

Plate 2-12. *Cross Section of the Monocot Stem of Zea mays (Corn)*

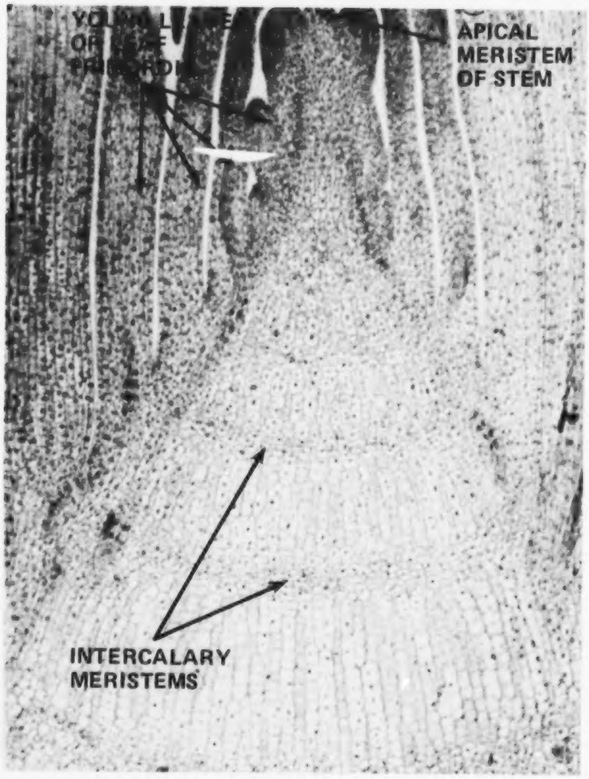
YOUNG LEAVES
OR LEAF
PRIMORDIA
APICAL
MERISTEM
OF STEM
INTERCALARY
MERISTEMS

Plate 2-13. *Longitudinal Section of the Shoot Tip of Spartina alterniflora (Smooth Cord Grass) Showing the Apical Meristem, Young Leaves and Intercalary Meristems*

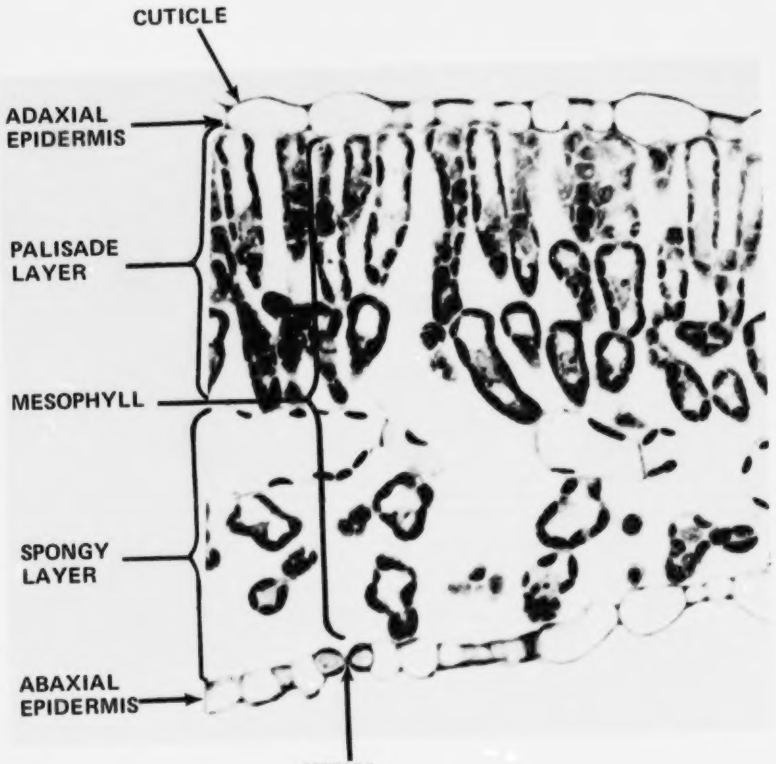
CUTICLE
ADAXIAL EPIDERMIS
PALISADE LAYER
MESOPHYLL
SPONGY LAYER
ABAXIAL EPIDERMIS
STOMA

Plate 2-14. *Cross Section of the Broad-leaved Leaf of Glycine max (Soybean)*

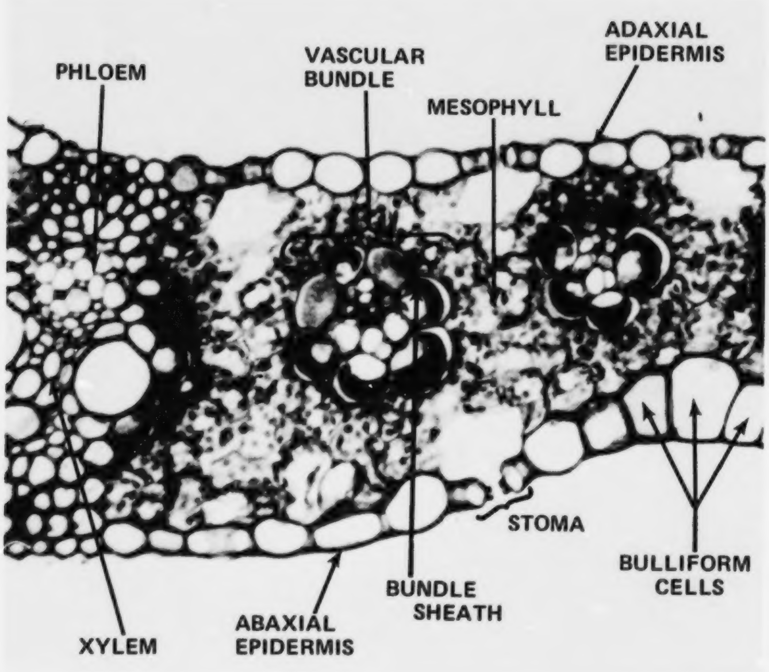
PHLOEM
VASCULAR BUNDLE
ADAXIAL EPIDERMIS
MESOPHYLL
STOMA
BULLIFORM CELLS
BUNDLE SHEATH
XYLEM
ABAXIAL EPIDERMIS

Plate 2-15. *Cross Section of the Narrow-leaved Leaf of Zea mays (Corn)*

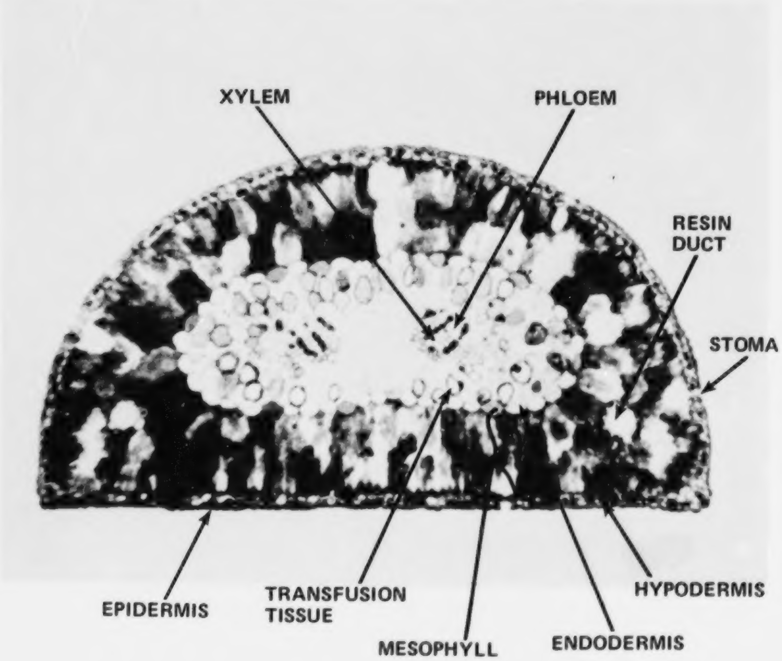
XYLEM
PHLOEM
RESIN DUCT
STOMA
EPIDERMIS
TRANSFUSION TISSUE
MESOPHYLL
ENDODERMIS
HYPODERMIS

Plate 2-16. *Cross Section of the Needle-like Leaf of Pinus sp. (Pine)*

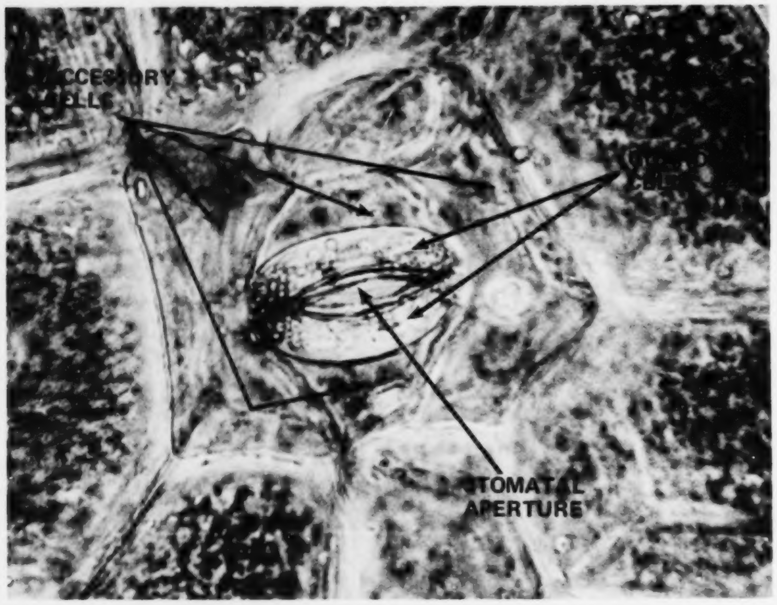
ACCESSORY CELLS
STOMATAL APERTURE

Plate 2-17. *Surface View of a Stoma of Zebrina sp. (Wandering Jew)*

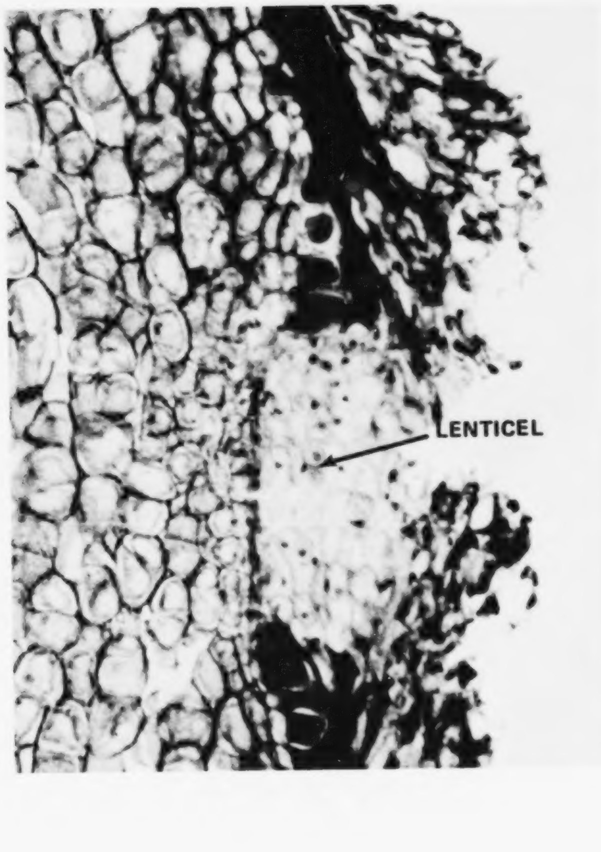

Plate 2-18. *Cross Section of the Outer Bark of Liriodendron tulipifera (Tulip Poplar) Showing a Lenticel*

Chapter 3

THE PHOTOCHEMICAL OXIDANTS

Introduction

In the early 1900's when a dearth of wood fuels forced Londoners to use coal as a domestic fuel, coal smoke, combined with fog, became a public nuisance which was to last many years and cause much suffering and many deaths. The term "smog" was coined then, a combination of the words smoke and fog. During the rapid industrial and urban expansion that followed World War II, air pollution episodes began to appear with increasing frequency in the larger urban areas of the world. It was only natural that people referred to this nuisance also as smog since there was general belief, even among the scientific community, that the basic problem was sulfur dioxide in coal smoke. However, during the 1950's the chemical constituents of this smog were identified, and it was discovered that this smog arose from a series of atmospheric reactions between hydrocarbons and oxides of nitrogen from automobile exhaust in the presence of sunlight. Among the products produced by the reactions are substances called "oxidants." Since sunlight was needed to initiate the reactions, the terms "photochemical smog," or more specifically "photochemical oxidants," are presently being used to differentiate it from the London-type smog.

Because of the complexity of this subject matter, this chapter has been divided into three sections, based on the three most important constituents from the standpoint of plant damage. The interrelationship of the three parts, however, and their relation to the total air pollution problem, should be kept in mind. The three phytotoxic constituents consist of *ozone* (O_3), the *peroxyacyl nitrates* (Pans), and *oxides of nitrogen.* Four peroxyacylnitrates have been identified: *peroxyacetyl nitrate* (PAN), *peroxypropionyl nitrate* (PPN), *peroxybutyryl nitrate* (PBN), and *peroxyisobutyryl nitrate* (P_{iso}BN). Although PPN is more toxic to vegetation than PAN, and PBN and P_{iso}BN are more toxic than PPN, only PAN has been studied extensively. PAN also appears to be the only one of this homologous series to be present in smog at concentrations high enough to cause injury and, therefore, will be the only member of the series discussed in this chapter.

SECTION 1. OZONE

Sources

Ozone is formed naturally by the action of sunlight on oxygen in the upper atmosphere (15,000 to 37,000 meters). Ozone can also be formed by electrical discharge during thunderstorms. These two natural sources, however, do not contribute significantly to the smog problem because: (1) the electrical energy available from the atmosphere is not adequate to form significant ozone concentrations over large urban areas, and (2) although ozone in the upper atmosphere can be transferred to the ground in the vicinity of jet streams and weather fronts, these concentrations are not believed to be high. The anthropogenic source of ozone is the principal cause of plant injury from this pollutant. Many pollutants arrive at the leaf surface in the same form in which they are emitted from smokestacks or automobile tailpipes. But photochemical oxidants are quite different. These compounds are called "secondary" pollutants, because they are formed in the air by the action of sunlight on primary air pollutants. Such reactions are chemical in nature, and are mediated by sunlight. Thus they are called "photochemical" reactions. Since some of the photochemical products formed are capable of "oxidizing" certain materials (such as nylon, rubber, plant tissue, etc.), they are called *photochemical oxidants*. The two most important photochemical oxidants from the standpoint of plant injury are *ozone* and *peroxyacetyl nitrate*.

The primary pollutants involved in the photochemical reactions producing ozone and PAN are hydrocarbons and oxides of nitrogen. Hydrocarbons arise during the incomplete combustion of petroleum products such as natural gas, gasoline, or oil. Although many industries emit hydrocarbons, *transportation sources account for more than half of the total emissions.*

The photochemical reactions that take place in an atmosphere containing oxygen and oxides of nitrogen without hydrocarbons can be generalized as shown in Figure 3-1. The oxides of nitrogen are broken down into nitric oxide (NO) and atomic oxygen (O). The atomic oxygen then reacts with the normal oxygen (O_2) of the air to form ozone (O_3). The O_3 may further react with the NO to form NO_2 and O_2, in a balanced, cyclic reaction.

However, if ozone (O_3) always reacted with NO to form NO_2, a steady-state system would exist with no ozone build-up. Of course, ozone does build up in polluted areas, because hydrocarbons enter into the reaction (Figure 3-2). It is postulated that the hydrocarbons disrupt the cyclic nature of the reaction by combining first with O_2 to form a peroxyacyl radical and then with NO_2 to form PAN. This removal of NO from the system allows for a build-up of ozone.

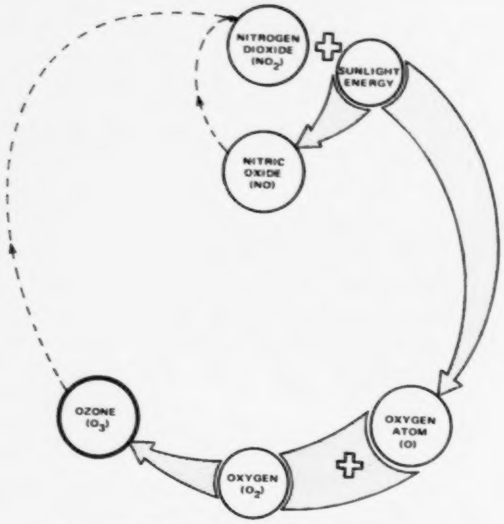
NITROGEN DIOXIDE (NO_2)
SUNLIGHT ENERGY
NITRIC OXIDE (NO)
OXYGEN ATOM (O)
OXYGEN (O_2)
OZONE (O_3)

Figure 3-1. *Atmospheric NO_2 Photolytic Cycle*

Source: *Air Quality Criteria for Photochemical Oxidants.* Washington, D.C.: Public Health Service, Department of Health, Education, and Welfare, 2-4, 1970.

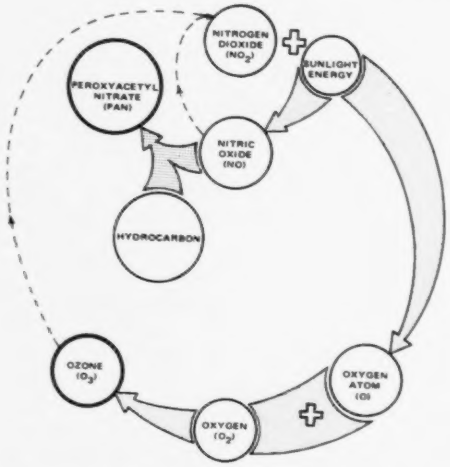
NITROGEN DIOXIDE (NO_2)
SUNLIGHT ENERGY
PEROXYACETYL NITRATE (PAN)
NITRIC OXIDE (NO)
HYDROCARBON
OXYGEN ATOM (O)
OXYGEN (O_2)
OZONE (O_3)

Figure 3-2. *Photolytic Cycle with Hydrocarbons*

Source: Adapted from: *Air Quality Criteria for Photochemical Oxidants.* Washington, D.C.: Public Health Service, Department of Health, Education, and Welfare, 2-7, 1970.

Atmospheric Chemistry

The photochemical reactions and subsequent high oxidant levels are most severe during inversions and stagnant weather. Normal background levels of ozone are 0.02 to 0.04 ppm* (39 to 78 $\mu g/m^3$),** but during inversions the concentrations of primary pollutants build up in the stagnant air, and the photochemical reaction causes an increase in the oxidant concentration. Levels of total oxidants (90 percent or more of which are ozone) exceeding 0.50 ppm (880 $\mu g/m^3$) have been recorded during such weather in Los Angeles. Peaks of 0.15 to 0.25 ppm (294 to 490 $\mu g/m^3$; the levels are for oxidants) are common in many urban areas during inversions. Such levels are capable of severely injuring sensitive plants.

Maximum oxidant levels often occur in mid to late summer (Figure 3-3). In this figure, the curve for Denver is probably more representative for the entire country than the curves for Los Angeles and Phoenix. Oxidant levels at various

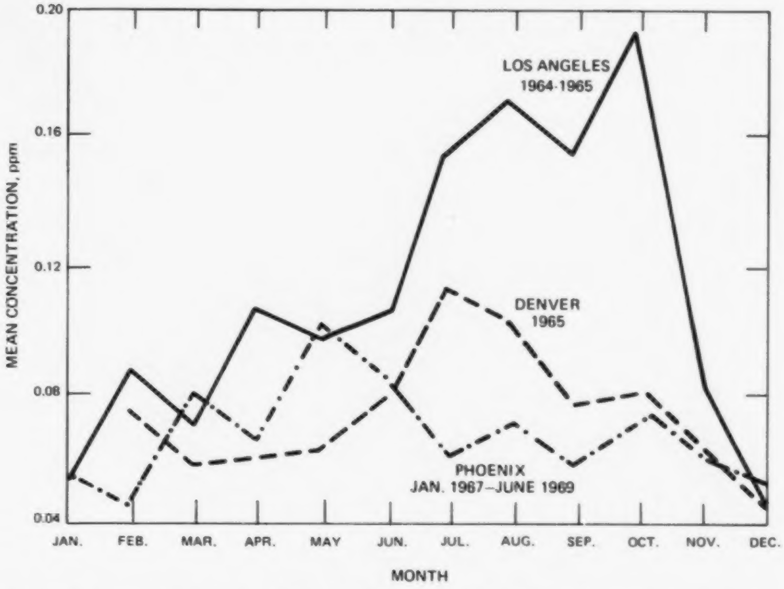

Figure 3-3. ***Monthly Variation of Mean Daily Maximum One-Hour Average Oxidant Concentrations for Three Selected Cities***

Source: ***Air Quality Criteria for Photochemical Oxidants.*** **Washington, D.C.: Public Health Service, Department of Health, Education, and Welfare, 3-8, 1970.**

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*ppm = parts per million, v/v

**$\mu g/m^3$ = micrograms of pollutant per cubic meter of air

NOTE: See Appendix A for explanation of conversions between ppm and $\mu g/m^3$.

times of day also show consistent patterns (Figures 3-4, 3-5, 3-6). Typically, oxidant concentrations are low during the night, begin to rise in the morning, peak in early afternoon, and then drop again as evening approaches.

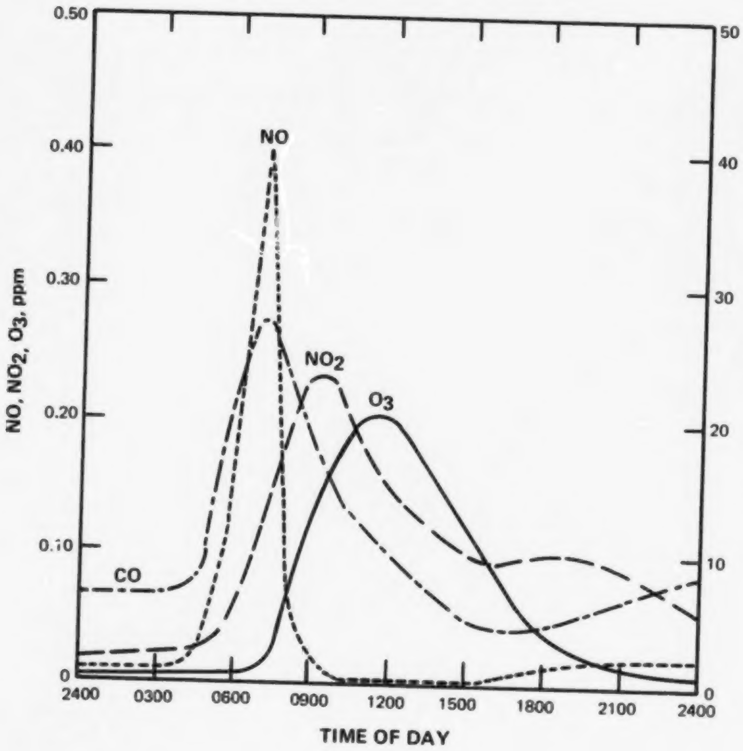

Figure 3-4. *Average Daily One-Hour Concentrations of Selected Pollutants in Los Angeles, July 19, 1965*

Source: *Air Quality Criteria for Nitrogen Oxides.* Washington, D.C.: Environmental Protection Agency, Air Pollution Control Office, 1971.

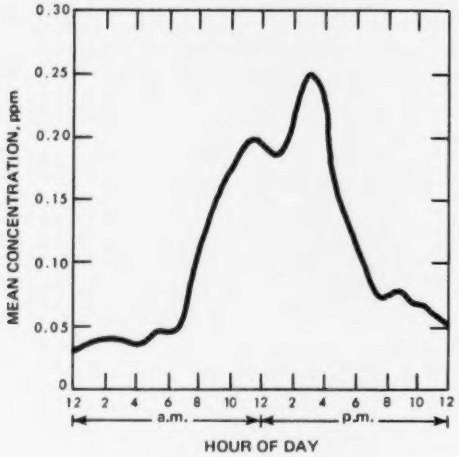

Figure 3-5.
Diurnal Variations of Mean Hourly Average Oxidant Concentrations in Philadelphia, August 6-8, 1966

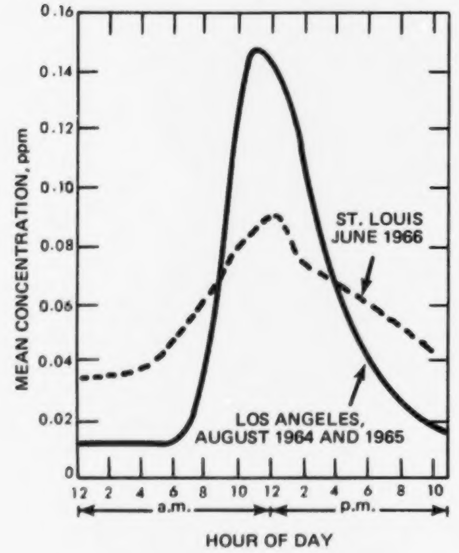

Figure 3-6.
Diurnal Variation of Mean Hourly Average Oxidant Concentrations in Los Angeles and St. Louis

Source: *Air Quality Criteria for Photochemical Oxidants.* Washington, D.C.: Public Health Service, Department of Health, Education, and Welfare, 3-8, 1970.

Monitoring Data

Long-term trends for oxidants are presented in Table 3-1 for four major U. S. cities. Although the maximum hourly averages have increased over the sampling period, there does not appear to be a uniform predictable pattern. There is likewise no pattern discernible if the available data are analyzed by number of days with at least one hourly average equal to or exceeding a given concentration. The reason for a lack of pattern may be related to the fact that photochemical pollutants are products of the weather in one sense. Since sunlight is required for ozone formation, the amount of cloudiness would affect concentrations, and cloudiness is variable from year to year.

Table 3-1
Data Showing Long-term Trends in
Oxidant Concentrations Recorded at CAMP Sites
from 1964 to 1967 in Four Large Urban Areas

City	*Year*	*Maximum hourly average, ppm*
Chicago	1964	0.13
	1965	0.13
	1966	0.19
	1967	0.16
Denver	1965	0.25
	1966	0.19
	1967	0.21
Philadelphia	1964	0.20
	1965	0.33
	1966	0.52
	1967	0.17
Washington, D.C.	1964	0.20
	1965	0.21
	1966	0.16
	1967	0.26

Source:
Adapted from: *Air Quality Criteria for Photochemical Oxidants.* Washington, D.C.: Public Health Service, Department of Health, Education, and Welfare, 3-4, 1970.

The oxidants formed over large urban areas may cause plant injury many miles away. For example, oxidants formed over the Los Angeles basin injure pines in the San Bernardino Mountains, 60 to 70 miles from the city's center, as the polluted air mass moves eastward. Recent studies by the Environmental Protection Agency (EPA) have suggested that high ozone concentrations in rural parts of Maryland and West Virginia may result from primary pollutants released from urban-industrial areas many miles away. As these precursor pollutants move eastward with the prevailing weather patterns, ozone is formed in the presence of sunlight, and the precursors are consumed, leaving fairly high ozone levels which are capable of causing plant injury in remote areas. Thus, it is important to consider ozone injury to vegetation in remote rural areas, as well as metropolitan areas.

Symptomatology

Ozone probably causes more plant damage in the U. S. than any other pollutant. Injury has been observed in the eastern, central, and western U. S., in rural as well as urban areas, and on a wide range of plants including leafy vegetables, grains, and conifers.

Ozone is taken into the leaf through the stomata during normal gas exchange. Once inside the leaf, the ozone (or possibly a secondary product) attacks the cells, probably first disrupting the cell membrane. As these structures break down, groups of cells collapse and symptoms appear on the leaf surface. If the leaf contains palisade mesophyll cells, as do most broad-leaved plants, ozone preferentially attacks these cells and the symptoms may be initially restricted to the upper leaf surface. On narrow-leaved plants, conifers, and broad-leaved plants in which the mesophyll is not differentiated into palisade and spongy mesophyll, cells usually near stomata are attacked, and symptoms may appear on either leaf surface.

In general, ozone injury develops initially at the tips of young leaves, and becomes more widespread over the leaf surface as the leaves mature. The oldest, mature leaves are usually tolerant to ozone, while the next-to-oldest are often sensitive, as on tobacco, although there are exceptions to this. Occasionally, older leaves may show severe ozone injury late in the season, possibly because they were exposed for a longer period of time, including their period of peak sensitivity. Many other factors such as plant age, nutrition, climatic factors, time of year, shading, etc. may affect symptom development to varying degrees.

Broad-leaved (Dicotyledonous) Plants

Usually, although not always, the first symptom of ozone injury on broad-leaved plants is a watersoaked appearance of the upper leaf surface (Plate 3-1). The leaf appears to be shiny, or oily, and if the tissue is injured, the watersoaked areas become dry and characteristic symptoms form. If the tissues are not irreversibly injured, the watersoaked look may disappear, and the leaf will recover. The most common ozone symptoms on broad-leaved plants are small *flecks* or *stipples* visible on the upper leaf surface.

The individual flecks, as seen with a hand lens, are small lesions of dead tissue, formed when groups of palisade cells die. The upper epidermal cells over the injured palisade cells also collapse. The cells in the affected area may die and become bleached. The individual flecks may be white, yellow, or tan (Plates 3-2 through 3-12). If the flecks are numerous, the entire leaf appears chlorotic (yellowish or pale green), or bronzed (Plates 3-13, 3-14). Premature defoliation often accompanies chlorosis or bronzing.

Acute injury. If a plant is extremely sensitive, or if a high dose of ozone is involved, the flecks may coalesce into larger lesions, occasionally visible on both leaf surfaces. Small veins may even be killed in this larger "bifacial" lesion (Plate 3-15). This severe symptom is observed in the field only on extremely sensitive plants, and is more commonly described for plants subjected to artificial exposures of ozone.

Stipples are small, dark, pigmented groups of palisade cells that appear as small dark dots visible on the upper leaf surface after exposure (Plates 3-16, 3-17, 3-18). The upper epidermis often remains uninjured, and the symptom is thus actually observed through the epidermal cells. Stipples may be red, reddish-brown, reddish-purple, or black. Veins and veinlets are uninjured, and the smaller veinlets actually form a boundary for the pigmented area, causing the lesion to be angular in shape. Stipples are best viewed with a hand lens, and, on dark green leaves, may be best observed when the leaf is held up to the light.

Chronic injury. Chronic injury is usually associated with vegetation exposed to relatively low concentrations of ozone for fairly long time periods. Depending upon the plant species, either the fleck or the stipple symptom type may be present on broad-leaved plants. Although the stipple type of symptom is generally regarded as a less severe reaction than the fleck type, the symptom produced is more often correlated with the *plant species* than with the concentration or duration of pollutant.

Symptom expression is often not intense in the case of chronic injury. Few symptoms may appear on the leaf surface. However, if the chronic injury is prolonged, the symptoms may gradually coalesce on the leaf surface, giving the injured leaf a bronzed or yellowed appearance. Bronzing or yellowing may be accompanied by premature defoliation. Reduced growth and/or reduced yield may also result from long-term exposures of vegetation to low levels of ozone. It has been shown that ozone can cause reduced crop yield in potatoes, grapes, citrus, and other plants.

Narrow-leaved (Monocotyledonous) Plants

Most narrow-leaved plants such as grass or grains, including corn, have photosynthetic cells and stomata on both surfaces of the leaves. Ozone injury on these plants thus appears on either leaf surface. The most common symptoms are small chlorotic spots or white flecks between the veins. Injury on sensitive plants appears as long white-to-yellow streaks between the veins, often being most severe at the bend in the leaf blade. A complete bleaching of the leaf blade (Plate 3-19) may also develop.

Coniferous Plants

The two most common ozone symptoms observed on conifers are "chlorotic mottle" and "tipburn." Chlorotic mottle develops as small patches of tissue in the needle are injured and become yellow. These islands of yellow tissue are surrounded by healthy green tissue, giving the needle a "mottled" appearance. Tipburn refers to the condition when the entire needle tip dies, becomes reddish-brown, and later gray as it weathers (Plate 3-20).

Acute injury. Acute ozone injury to most coniferous species is usually observed as tipburn of the current season's needles. Chlorotic mottle may accompany the tipburn in some cases. Previous seasons' needles are often not affected by acute exposures, nor are the needles of plants exposed in seasons other than spring and early summer.

Chronic injury. The chlorotic mottle symptom is a common expression of chronic ozone injury on individual needles of many coniferous species. However, needles of certain species such as eastern white pine may also exhibit tipburn after chronic exposure to ozone.

In general, chronic ozone injury to conifers is manifested as reduced growth. Symptoms include a reduction in both terminal and diameter growth, loss of all but the current season's needles, reduction in amount and size of the needles, deterioration of the fibrous root system, and a gradual decline and sometimes death of the tree (Plate 3-21).

In addition, chronic injury may result in a conifer's inability to withstand extremes in environmental factors such as drought, or secondary parasites such as root-rot fungi or bark beetles. Cone production may also be impaired by chronic ozone injury.

Symptoms of Common Disorders

Several specific disorders commonly observed in the field are caused by ozone. A knowledge of these disorders, since they are common, is valuable to the diagnostician. A brief description of each follows:

Grape Stipple. This disorder was the first major plant problem diagnosed as being caused by ozone. Although first observed in southern California, it is now thought to be widespread in the East. The syndrome includes leaf bronzing, yellowing, premature senescence and leaf fall. Symptoms first appear on the upper surface of young leaves early in the season as brown-to-black stipples bounded by the smallest veinlets. The small lesions often coalesce to give rise to larger stipples (Plate 3-16). As the season progresses, the older leaves become bronzed and fall prematurely. Grape yields may be drastically reduced.

Weather Fleck of Tobacco. This problem has been severe on sensitive varieties of tobacco, especially cigar wrapper varieties from southern Canada to southeastern U. S. The primary symptom is the presence of white, light gray, or tan flecks on the upper leaf surface of fully expanded leaves (Plate 3-2). The symptoms are often confined by the smallest veins, thus forming lesions angular in shape. On sensitive varieties such as Bel W-3 the lesion may go completely through the leaf and be visible on the lower leaf surface (i.e., it becomes bifacial). Premature senescence, including yellowing, may occur in all tobacco species.

Onion Tipburn or Blight. This problem has occurred sporadically over the years in many widespread areas of the country. The symptom consists of typical ozone flecking and tissue breakdown, followed by death and browning of the onion leaf tip (Plate 3-22).

Emergence Tipburn of Eastern White Pine. Emergence tipburn, historically known as "white pine blight," has been observed on scattered trees throughout the natural range of white pine since the early 1900's (Plate 3-23). The problem is characterized by a dying of the tips of young elongating needles in June or

July. The injured tissue is reddish-brown at first, later turning brown (Plates 3-20, 3-21). As the dead tissue weathers, it turns gray and the needle tip may break off.

Ozone Needle Mottle of Ponderosa Pine. This disorder is widespread in many areas of California, but principally in the San Bernardino Mountains of southern California, where it is caused by ozone formed over Los Angeles drifting eastward. The earliest symptoms on sensitive trees are small, chlorotic flecks on the older needles. The chlorosis and yellowing become more severe, the needles become yellow, then brown and finally drop prematurely. Eventually, all but the current year's needles are lost, contrasted with the normal three-to-five years of needles present on resistant trees. Thus, the branches have a tufted appearance, being bare except for the current year's needles. In severe cases, even the current needles begin to die, then the branches die back and gradually the entire tree succumbs (Plate 3-21).

Factors Affecting Plant Response

Genetics

Genetically, plants vary considerably in their response to ozone. Certain species are quite sensitive, while others are very tolerant. Even within the same species, different cultivars may vary greatly in sensitivity. For example, the Bel W-3 cultivar of tobacco is extremely sensitive to ozone, whereas the Bel B is fairly tolerant. Species or cultivars of green beans, azaleas, radish, tomato, safflower, poplars, soybeans, petunia, and others have all demonstrated this variation in response within the same species.

Environmental Factors

Many environmental factors influence the sensitivity of plants to ozone, including climatic parameters such as light, temperature, and humidity. In addition, various soil features such as moisture, texture, and nutrition also affect plant response. Some of these variables influence sensitivity simply by altering stomatal opening, thereby influencing gas exchange and, at the same time, pollutant uptake. The influence of other variables is more subtle, possibly affecting internal physiological mechanisms which regulate sensitive membranes, repair mechanisms, and other internal functions of the plant.

Temperature. In general, plants grown at warm temperatures (i.e., 27 to 32°C) prior to ozone exposure are more sensitive to ozone than are those maintained at cooler temperatures (i.e., 10 to 16°C) before a fumigation. However, *during* an ozone fumigation, an inverse relationship between the amount of tissue injury and exposure temperature is observed. More injury occurs at lower temperatures than at higher temperatures. This indicates that maximum sensitivity to ozone, in terms of temperature, occurs when plants are growing under warm conditions, and then exposed to ozone in relatively cool air.

Humidity. Atmospheric humidity is usually directly related to ozone sensitivity. Plants grown and/or exposed at fairly high relative humidities (i.e., 80 to 90%) are more sensitive than those grown and/or exposed at low humidities (i.e., 50 to 60%). Plants growing in parts of the country having low humidities, such as the arid Southwest, are probably more tolerant to ozone and other air pollutants than are plants grown in the more humid Northeast.

Light. Light also influences the sensitivity of vegetation to ozone. Plants grown at low light intensities (i.e., 1000 to 2000 footcandles) prior to ozone exposure are more sensitive than are plants grown at higher intensities (i.e., 2000 to 3000 footcandles). However, *during* an exposure, plants exposed at higher intensities show more injury.

The amount of light during a day (photoperiod) also affects sensitivity. For example, tobacco and pinto bean plants are more sensitive when grown under an eight-hour photoperiod than under one of 16 hours. Also, plants are generally more sensitive at mid-day than in early morning or late afternoon, and least sensitive at night (during which time many species have closed stomata). The influence of light on ozone sensitivity is of special practical significance in commercial greenhouses, where light intensity and duration are often varied to induce growth, flowering, or fruiting.

Soil. Edaphic (soil) factors affecting the underground portion of a plant may have a profound effect on the ozone sensitivity of the above-ground portion. Moisture-stressed plants are usually more tolerant to ozone than are those growing under ample soil-moisture conditions. Presumably, the moisture stress induces closure of the stomata, preventing pollutant uptake. The soil texture (relative amounts of sand, silt, and clay) may also affect sensitivity. Plants grown in light, well-aerated soils are more sensitive than those in heavy clay soils. Soil moisture and aeration interrelationships are probably involved. The exact influence of soil nutrition on sensitivity is not clear, but in general, plants growing with optimal nutrients for growth are more sensitive to ozone than are those growing at nutrient deficiencies or excesses.

Injury Threshold Doses

Pollutant concentration and the length of the exposure period are collectively referred to as "dose," and the lowest dose that produces an effect is termed a "threshold dose." Because of the interrelationship of concentration and time, there is no single threshold dose for an effect to occur. For example, leaf injury on a given plant or set of plants exposed to .10 ppm (196 $\mu g/m^3$) O_3 might occur after only one hour, but when exposed to .05 ppm (98 $\mu g/m^3$) O_3 it might require an exposure of up to 10 hours to produce leaf injury.

The threshold dose of ozone that causes injury varies tremendously among species, and even among cultivars within the same species. However, the most sensitive species of various types of plants are all injured by approximately the same threshold concentration of ozone. For example, certain varieties of sweet corn and oats (monocots) are injured by exposure to 0.10 to 0.12 ppm (196 to 235 $\mu g/m^3$) ozone for two hours. Bel W-3 tobacco (a dicot), one of the most ozone-sensitive plants, shows leaf injury after exposure to 0.05 ppm (98 $\mu g/m^3$) for four hours. Selected, sensitive strains of eastern white pine (conifer) have been injured by 0.07 ppm (137 $\mu g/m^3$) for four hours.

Each of the above species or strains is extremely sensitive to ozone under laboratory conditions. Therefore, 0.05 to 0.10 ppm (98 to 196 $\mu g/m^3$) ozone for two to four hours probably represents the absolute threshold dose for sensitive species under conditions approximating laboratory conditions. Other varieties or strains of the same species are much more tolerant. Table 3-2 illustrates projected ozone concentrations that will produce, for short term exposures, threshold injury to vegetation grown under sensitive conditions.

Table 3-2

Threshold Doses of Ozone Needed to Cause Injury to Plants Grown Under Sensitive Conditions

Time (hours)	*Ozone concentration needed to cause injury*		
	Sensitive	*Intermediate*	*Tolerant*
0.5	.15-.30 ppm*	.25-.60 ppm	< .50 ppm
1.0	.10-.25	.20-.40	< .35
2.0	.07-.20	.15-.30	< .25
4.0	.05-.15	.10-.25	< .20
8.0	.03-.10	.08-.20	< .15

*1 ppm = 1,960 $\mu g/m^3$ at 25°C.

Source:
Heggestad, H. E. & Heck, W. W. Nature, Extent, and Variations of Plant Response to Air Pollutants. *Advances in Agronomy*, 23:111-145, 1971.

Air Quality Standards

The current air quality standard is 0.08 ppm (157 $\mu g/m^3$) ozone for one hour not to be exceeded more than once a year. This concentration is frequently exceeded in urban areas. Recent reports have shown this level is often exceeded in rural areas as well.

Only extremely sensitive plants growing under the most ideal conditions are injured after exposure to 0.05 to 0.10 ppm (98 to 196 $\mu g/m^3$) ozone for three to four hours. Such ideal growing conditions rarely occur in the field. It would be unlikely, therefore, that injury from ozone would occur if the standard is not exceeded. However, in nature, plants are often exposed to low levels of ozone many times throughout a growing season. Also, other air pollutants are often present in the ambient atmosphere. The chronic exposure of highly sensitive plants to low levels of ozone or to multiple pollutants simultaneously or sequentially, may result in slight vegetation injury when the ozone level is at or near the secondary air quality standard.

Relative Sensitivity of Plants to Ozone

Table 3-3 shows the relative sensitivity of various plants to ozone.

Table 3-3

Relative Sensitivity of Various Plants to Ozone*

Crops and Weeds

Sensitive	*Intermediate*	*Tolerant*
Alfalfa	Cabbage	Beet
Barley	Carrot	Cotton
Bean	Corn, Field	Descurainia
Buckwheat	Cowpea	Jerusalem cherry
Citrus	Cucumber	Lamb's-quarters
Clover, Red	Endive	Lettuce
Corn, Sweet	Hypericum	Mint
Grape	Parsley	Piggy-back plant
Grass, Bent	Parsnip	Rice
Grass, Brome	Pea	Strawberry
Grass, Crab	Peanut	Sweet Potato
Grass, Orchard	Pepper	
Muskmelon	Sorghum	
Oat	Stevia	
Onion	Timothy	
Potato	Turnip	
Radish		
Rye		
Safflower		
Smartweed		
Soybean		
Spinach		
Tobacco		
Tomato		
Wheat		

Ornamentals and Trees

Sensitive	*Intermediate*	*Tolerant*
Ash, Green	Alder	Apricot
Ash, White	Apple, Crab	Arborvitae
Aspen, Quaking	Apricot, Chinese	Azalea, Chinese
Azalea, Campfire	Begonia	Avocado
Azalea, Hino	Boxelder	Beech, European
Azalea, Korean	Carnation	Birch, European White
Azalea, Snow	Catalpa	Box, Japanese
Bridalwreath	Cedar, Incense	Dogwood, Gray
Browallia	Cherry, Lambert	Dogwood, White

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*Absence of any species from this or other sensitivity lists does not indicate that it is tolerant – only that it has probably not been studied. It should also be kept in mind that differences in tolerance exist among cultivars of these species.

Table 3-3 (Continued)

Relative Sensitivity of Various Plants to Ozone

Ornamentals and Trees (Continued)

Sensitive	*Intermediate*	*Tolerant*
Cherry, Bing	Chrysanthemum	Euonymus, Dwarf Winged
Coleus	Elm, Chinese	Fir, Balsam
Cotoneaster, Rock	Fir, White	Fir, Douglas
Cotoneaster, Spreading	Fir, Big-Cone Douglas	Fir, White
Grape, Concord	Forsythia, Lynwood Gold	Firethorne, Laland's
Lilac, Chinese	Grape, Thompson Seedless	Fuchsia
Lilac, Common	Gum, Sweet	Geranium
Locust, Honey	Honeysuckle, Blue-leaf	Gladiolus
Mountain Ash, European	Larch, European	Gloxinia
Oak, Gambel	Larch, Japanese	Gum, Black
Oak, White	Mock Orange, Sweet	Hemlock, Eastern
Petunia	Oak, Black	Holly, American (Male)
Pine, Austrian	Oak, Pin	Holly, American (Female)
Pine, Coulter	Oak, Scarlet	Holly, Hetz's Japanese
Pine, Eastern White	Pine, Knobcone	Impatiens
Pine, Jack	Pine, Lodgepole	Ivy, English
Pine, Jeffery	Pine, Pitch	Juniper, Western
Pine, Monterey	Pine, Scotch	Lemon
Pine, Ponderosa	Pine, Sugar	Laurel, Mountain
Pine, Virginia	Pine, Torrey	Linden, American
Poplar, Hybrid	Poinsettia	Linden, Little-leaf
Poplar, Tulip	Privet, Common	Locust, Black
Privet, Londense	Redbud, Eastern	Maple, Norway
Snowberry	Rhododendron, Catawbiense Album	Maple, Sugar
Sumac	Rhododendron, Nova Zembia	Maple, Red
Sycamore, American	Rhododendron, Roseum Elegans	Marigold
Tree of Heaven	Silverberry	Mimosa
Walnut, English	Viburnum, Linden	Oak, Bur
	Viburnum, Tea	Oak, English
	Walnut, English	Oak, Northern Red
	Willow, Weeping	Oak, Shingle
		Pachysandra
		Pagoda, Japanese
		Peach
		Pear, Bartlett
		Periwinkle
		Pieris, Japanese
		Pine, Digger
		Pine, Singleleaf Pinyon
		Pine, Red
		Privet, Amur North
		Redwood
		Rhododendron, Carolina

Table 3-3 (Continued)
Relative Sensitivity of Various Plants to Ozone

Ornamentals and Trees (Continued)		
Sensitive	*Intermediate*	*Tolerant*
		Sequoia, Giant
		Snapdragon
		Spirea
		Spruce, Black Hills
		Spruce, Colorado Blue
		Spruce, Norway
		Spruce, White
		Viburnum, Koreanspice
		Viburnum
		Virginia-Creeper
		Walnut, Black
		Yew, Dense
		Yew, Hatfield's Pyramidal
		Zinnia

Leaf Tissue Analysis

Ozone is a very strong oxidant. It breaks down immediately upon contact with any oxidizable substance, including, of course, plant tissues. It leaves no residues which could be analyzed by chemical means to confirm diagnosis.

SECTION 2. PAN

Introduction

PAN (peroxyacetyl nitrate) injury to plants is not as widely distributed as ozone injury; it has been a major problem primarily on vegetables grown in the Los Angeles basin and has occurred to a lesser degree in other large western cities. PAN concentration reported in urban areas of California ranged from 0.01 to 0.02 ppm (49 to 99 $\mu g/m^3$) on average days, with a maximum concentration reaching to 0.05 ppm (247 $\mu g/m^3$) for one hour. In the East, suspected PAN injury has been described on vegetables and tomatoes in New Jersey and southern Canada. However, the actual occurrence of PAN and subsequent plant injury in the East has not been proven, and mimicking symptoms must carefully be considered when diagnosing PAN injury.

Since the *Sources, Atmospheric Chemistry,* and *Monitoring Data* for PAN are the same as for ozone, the reader is advised to see pages 3-2 to 3-8 for this information.

Symptomatology

PAN, like other gaseous pollutants, is taken into the leaf through the stomata during normal gas exchange. Once inside the leaf, PAN attacks preferentially the spongy mesophyll cells but may also attack other tissues depending on the species. Thus, on many broad-leaved plants, the symptoms are visible predominantly on the lower leaf surface. On monocots, such as corn, the symptoms are present on both leaf surfaces. Injury from ambient levels of PAN has not been described on trees.

Young, rapidly growing plants are generally more sensitive to PAN than are older plants of the same species. For example, young tomato or petunia plants with only three or four leaves are more sensitive than comparable plants with six or eight leaves. On an individual plant, young, rapidly expanding leaves are most sensitive. PAN injury appears on the apex of younger leaves, as a diffuse band on intermediate-aged leaves, and at the base of older (but sensitive) leaves. Mature leaves are tolerant to PAN. Plate 3-24 shows a comparison of ozone and PAN injury on alfalfa.

Broad-leaved (Dicotyledonous) Plants

Herbaceous broad-leaved plants such as table beet, sugar beet, lettuce, bean, and spinach are among the plants most sensitive to PAN. The earliest symptom of PAN injury on these plants is a watersoaking or shininess of the lower leaf surface. As the injury progresses, the spongy mesophyll cells, especially those near the stomata, collapse and air pockets take their place. The air pockets give the undersurface of the leaf a glazed or silvery appearance – the "classic" symptom of PAN injury (Plates 3-25, 3-26). After two or three days, the glazed surface may become bronzed (Plates 3-27, 3-28).

Some plants such as petunia do not develop the classic "silvering" symptom.

Instead, the dominant symptom is a bifacial necrosis, very similar to injury caused by some other pollutants (Plate 3-29).

Banding. Banding is an important symptom associated with PAN injury. For example, when leaflets of compound leaves such as tomato or potato are exposed to PAN, symptoms appear at the tip of the youngest susceptible leaf. The next-oldest leaf is usually injured near the base of the terminal leaflet and at the apex of the first two terminal lateral leaflets. The third-oldest susceptible leaf is subsequently injured at the base of the first two lateral leaflets and at the apex of the second pair of lateral leaflets, while the more mature terminal leaflet remains uninjured. As the leaf tissue grows and matures, the injured areas appear as bands. A second exposure to PAN will produce a second set of bands, separated from the first set by healthy leaf tissue. With several successive exposures to PAN, injury will develop over the entire leaf. On certain plants such as petunia, the injured tissue in the band area will form an indented or "pinched" area at the edge of the leaf because part of the expanding leaf has been killed.

Narrow-leaved (Monocotyledonous) Plants

Many monocots such as grasses and grains are injured by PAN. The symptom appears as distinct bands across the leaf blade (each band is 2 or 3 to 7 or 8 millimeters in width). The tissue in the band may be chlorotic (yellow) if the injury is slight, or totally collapsed and bleached out if the injury is severe (Plate 3-30). Glazing and bronzing seldom appear.

A single exposure to PAN will produce a band near the tip of a young leaf, near the middle of the next-oldest leaf, and at the base of the next-oldest leaf. As the tissue in the band collapses, the entire leaf blade folds downward at that point. The tip of the blade retains its normal green color for several days or longer. Further exposure over various time intervals will produce more bands along the leaf, with green tissue separating the bands.

Coniferous Plants

PAN injury has not been described on conifers.

PAN-Mimicking Symptoms

Frost and Sunscald. Perhaps the most common agent capable of causing PAN-like symptoms is frost, which causes an undersurface glazing or bronzing, especially on broad-leaved plants. However, frost does not attack the same specific leaf tissue *by age,* as PAN does. Frost injury is also usually most apparent on the upper leaf surface of many plants. Banding is not associated with frost injury, although cold injury may cause such banding on monocots. Sunscald on leaves turned over by the wind may also resemble PAN injury.

Insects. Certain insect infestations such as mites, leaf hoppers, and thrips may occasionally cause a lower-surface whitening or glazing. However, banding is not usually associated with such infestations, and the presence of the insect is usually evident.

Other Pollutants. Under certain conditions, hydrogen chloride may cause under-surface glazing and silvering. Hydrogen chloride does not usually cause banding, and it injures more mature tissue than does PAN. Further, hydrogen chloride injury is usually a local problem adjacent to a recognizable source. Other pollutants, including ozone, sulfur dioxide, and a combination of these two, can cause under-surface glazing as well.

Factors Affecting Plant Response

As with ozone, both inherent and environmental factors affect plant sensitivity to PAN. Some plants, such as petunia, romaine lettuce, and pinto bean, are inherently sensitive to PAN, while others such as pines, maples, and many other tree species are very tolerant. However, as with other pollutants, differences in PAN sensitivity exist among varieties of the same species.

Many environmental factors influence the sensitivity of vegetation to PAN. Light is a particularly important factor in regulating PAN sensitivity. High light intensity significantly increases sensitivity of plant tissue to PAN, and light must be present before, during, and after exposure for visible symptoms to develop. Therefore, in the field, plants exposed to PAN at mid-day on bright, sunny days would probably show the maximum amount of injury.

Any factor that influences stomatal opening during exposure to PAN influences sensitivity. More tissue is usually injured on plants exposed to PAN at high humidity than at low humidity. Likewise, soil moisture levels also affect sensitivity. Plants growing at optimum soil moisture levels exhibit more PAN injury when exposed than do those growing at soil moisture deficits, probably because of resulting stomatal closure.

Unlike ozone, the percent of leaf tissue injured by PAN is directly related to exposure temperature. That is, plants exposed to PAN at higher temperatures (27 to 32°C) are more severely injured than those plants exposed at lower temperatures (10 to 16°C).

Injury Threshold Doses

Plants are injured with much lower dosages of PAN than with ozone. Sensitive plants exhibit leaf injury after exposure to 0.01 to 0.02 ppm (49 to 99 $\mu g/m^3$) for four hours. For comparison purposes, concentrations of 0.02 to 0.03 ppm (99 to 148 $\mu g/m^3$) PAN occur frequently at Riverside, California, and a maximum peak of nearly 0.06 ppm (297 $\mu g/m^3$) was measured there for one two-hour period. Peaks as high as 0.05 ppm (247 $\mu g/m^3$) were reported at Salt Lake City, and a maximum concentration of 0.21 ppm (1,039 $\mu g/m^3$) was once measured in downtown Los Angeles. These concentrations are all substantially above the injury threshold dose of 0.01 to 0.02 ppm (49 to 99 $\mu g/m^3$). Such levels of PAN, if of sufficient duration, can cause widespread plant injury, especially on sensitive leafy vegetables.

Air Quality Standards

Air quality standards have not been formulated specifically for PAN.

Relative Sensitivity of Plants to PAN

Table 3-4 shows the relative sensitivity of various plants to PAN.

Table 3-4
Relative Sensitivity of Various Plants to PAN

Crops and Weeds

Sensitive	*Intermediate*	*Tolerant*
Bean	Alfalfa	Bean, Lima
Celery	Barley	Broccoli
Chard, Swiss	Beet, Sugar	Cabbage
Chickweed	Beet, Table	Cauliflower
Clover	Carrot	Corn
Endive	Cheeseweed	Cotton
Grass, Annual Blue	Dock, Sour	Cucumber
Ground-cherry	Lamb's-quarters	Onion
Jimson-weed	Soybean	Radish
Lettuce	Spinach	Rhubarb
Mustard	Tobacco	Sorghum
Nettle, Littleleaf	Wheat	Squash
Oat		Strawberry
Pepper		
Pigweed		
Tomato		
Wild-oat		

Ornamentals and Trees*

Sensitive	*Tolerant*	
Aster	Apple	Lily
Dahlia	Arborvitae	Locust, Honey
Fuchsia	Ash, Green	Maple, Norway
Mimulus	Ash, White	Maple, Silver
Mint	Azalea	Maple, Sugar
Petunia	Basswood	Mountain-Ash, American
Primrose	Begonia	Narcissus
Ranunculus	Birch, European White	Oak, English
Sweet-Basil	Bromiliads	Oak, Northern Red
	Cactus	Oak, Pin
	Calendula	Oak, White
	Camellia	Orchids
	Carnation	Periwinkle
	Chrysanthemum	Pine, Austrian
	Coleus	Pine, Eastern White
	Cyclamen	Pine, Pitch
	Dogwood	Pine, Red
	Fir, Balsam	Pine, Scotch
	Fir, Douglas	Pine, Virginia
	Fir, White	Poplar, Hybrid
	Gum, Sweet	Poplar, Tulip
	Hemlock, Eastern	Snapdragon
	Ivy	Spruce, Black Hills
	Larch, European	Spruce, Blue
	Larch, Japanese	Spruce, Norway
	Lilac, Common	Spruce, White

*None reported to be intermediate in sensitivity

Leaf Tissue Analysis

Like ozone, PAN breaks down when it comes in contact with plant tissues. No chemical test of injured plant tissue has been described to confirm diagnosis.

SECTION 3. OXIDES OF NITROGEN

Introduction

Oxides of nitrogen include nitric oxide (NO), nitrogen dioxide (NO_2), nitrous oxide (N_2O), nitrogen sesquioxide (N_2O_3), nitrogen tetraoxide (N_2O_4), and nitrogen pentoxide (N_2O_5). Of these, two are important as air pollutants: NO and NO_2. Although all oxides-of-nitrogen-induced plant injury is believed to result from NO_2 rather than NO, the latter will also be discussed in this section, because of its close association with NO_2 in atmospheric reactions. The two oxides of nitrogen, NO and NO_2, will be referred to collectively as NO_x.

Since the early 1950's, it has been known that nitrogen oxides are associated with photochemical smog, but their effects have not been studied as thoroughly as those of other smog components. Nevertheless, it has been established that NO_2 in the atmosphere can adversely affect plants, either directly or indirectly. Direct effects result mainly from NO_2 exposures and occur in localized areas. Indirect effects result from the key role of oxides of nitrogen in forming highly phytotoxic photochemical oxidants, such as ozone and PAN. At the present time, this indirect effect has a greater influence on vegetation than direct assaults of NO_2.

On an equivalent-concentration basis, oxides of nitrogen are less phytotoxic than most of the other major air pollutants discussed in this manual (ozone, PAN, sulfur dioxide, and fluoride). Consequently, the direct effects of NO_x on plants usually occur in localized areas, and are not too common. However, this situation will change if the existing trend toward increasing NO_x emissions continues.

Sources

The sources of NO_x in the atmosphere are both natural and man-made. On a worldwide basis, bacteria produce about ten times more NO than the combined total of NO and NO_2 emissions related to man's activities. According to Environmental Protection Agency estimates for 1972, industrial processes accounted for only 3% of man-related NO_x emissions, and more than 90% of this came from the combustion of fuels. Nearly 22 million tons of NO_x were emitted, of which about 56% was from stationary sources that burned coal, oil, natural gas, or other fuels. Nearly 40% of the total NO_x emissions were from combustion of motor-vehicle fuels, or related to other forms of transportation.

Although industrial processes comprise only a small proportion of the total NO_x released to the atmosphere, these sources provide a disproportionately greater direct threat to agriculture. Localized episodes of vegetation injury due to accidental NO_x releases or spills usually occur in areas where nitric acid is manufactured or used, or near electroplating, engraving, welding, or metal-cleaning operations. The manufacture of munitions, detonation of explosives, or release of rocket propellents may also result in NO_x emissions.

Atmospheric Chemistry

The most important features of oxides-of-nitrogen atmospheric reactions are those related to the generation of O_3 and other photochemical oxidants. The initial step in this process is the photodissociation of NO_2: ultraviolet light energy disrupts chemical bonds in NO_2 to form NO and atomic oxygen (O). Atomic oxygen reacts with atmospheric oxygen (O_2) to form O_3. (Some atomic oxygen may react with hydrocarbons in the air and produce other oxidant-type pollutants.) The NO produced by the photodissociation of NO_2 reacts with O_3 to form NO_2 and O_2. The process is essentially cyclic; it is rapid; and, with the exception of minor secondary or intermediate reactions, it occurs as shown in Figure 3-1.

As shown in Figure 3-4, significant concentrations of O_3 cannot accumulate until the NO concentration has all but disappeared from the atmosphere through reactions of NO with hydrocarbon compounds, resulting in the sequential occurrence of peak concentrations of NO, NO_2, and O_3. (Injury to plants where accidental releases or spills of NO occur are probably due to NO_2 rather than NO, because NO is rapidly oxidized to NO_2 in the atmosphere.)

Monitoring Data

The amount of nitrogen oxides in the atmosphere varies greatly with location and time. The potential for high NO_x concentrations is much greater in urban or industrial areas than in suburban or rural environments. At any location, the combined effects of man's activities and climatic and meteorological conditions result in diurnal, weekly, and annual influences on ambient NO_x concentrations.

Diurnal patterns of NO_x levels in urban areas are similar to those shown in Figure 3-4. Pre-dawn concentrations of NO and NO_2 are relatively stable. As human activity, especially automobile traffic, increases after sunrise, the concentration of NO increases rapidly. Later, as NO is converted to NO_2, the NO_2 concentration increases and NO decreases. When the NO concentration is lowest, O_3 and other photochemical oxidants begin to accumulate. In the late afternoon, solar radiation decreases, and the NO resulting from evening automobile traffic is not accumulated or converted to other oxidants, rather, it rapidly reacts with O_3 to form NO_2. This reaction continues into the night until the O_3 supply is consumed.

The weekly pattern of ambient NO_x concentrations is a composite of the diurnal patterns described above. The difference between daily and weekly patterns is undoubtedly due to variations in automobile traffic, because there is little day-to-day variability except on weekends and holidays. The difference in the average NO concentrations in Chicago over a two-year period for weekdays and weekends, shown in Figure 3-7, clearly illustrates this phenomenon.

The seasonal patterns for ambient NO and NO_2 concentrations are dissimilar. The average ambient concentrations of NO are highest in the winter months, when solar radiation for photochemical reactions is limited and NO emissions from power generation and space heating are increased. The average monthly ambient NO_2 concentrations, on the other hand, are relatively constant throughout the year (Figure 3-8). The proportion of NO_2 to the total NO_x concentration is greatest during the summer months, when the rate of the conversion of NO to NO_2 is greatest.

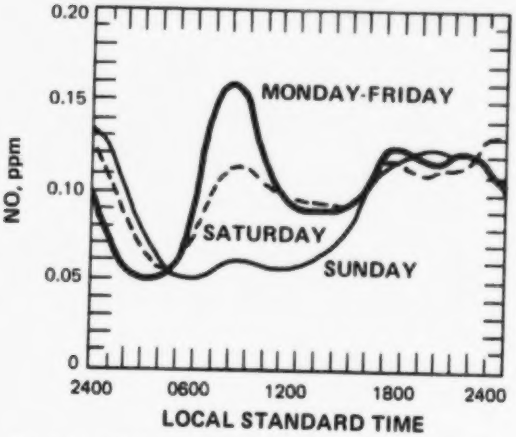

Figure 3-7. *Weekday and Weekend One-Hour Average NO Levels in Chicago, 1962 through 1964*

Source: *The Automobile and Air Pollution, A Program for Progress: Part II.* Washington, D.C.: Department of Commerce, December 1967.

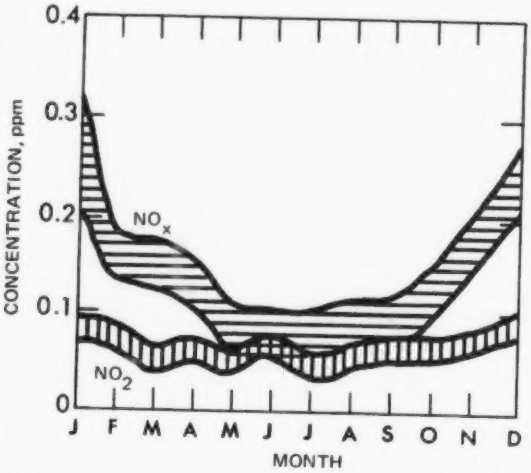

Figure 3-8. *Average and Standard Deviation of Daily Maximum Oxides of Nitrogen and Nitrogen Dioxide Concentrations for Seven Locations in Los Angeles Basin, 1962*

Source: Schuck, E. A., Pitts, Jr., J. N. & Wan, J. K. S. Relationships between certain meteorological factors and photochemical smog. *Air and Water Pollution,* 10:689-711, 1966.

Increased combustion of fuels has resulted in increased NO_x in the atmosphere. The nationwide scope of these increases is illustrated by the rates at which the annual average NO_x concentrations increased in the five major U.S. cities included in CAMP (Continuous Air Monitoring Program). Measurements covered 1962-1971, and the results are shown in Figure 3-9. Although most NO_x exposures sufficient to induce symptoms on vegetation occur as the result of localized accidental releases, metabolic effects that may affect plant growth and yield can be induced by extended exposures to much lower concentrations. The general increase in ambient NO_x concentrations, if continued, may pose serious problems for agriculture in the future.

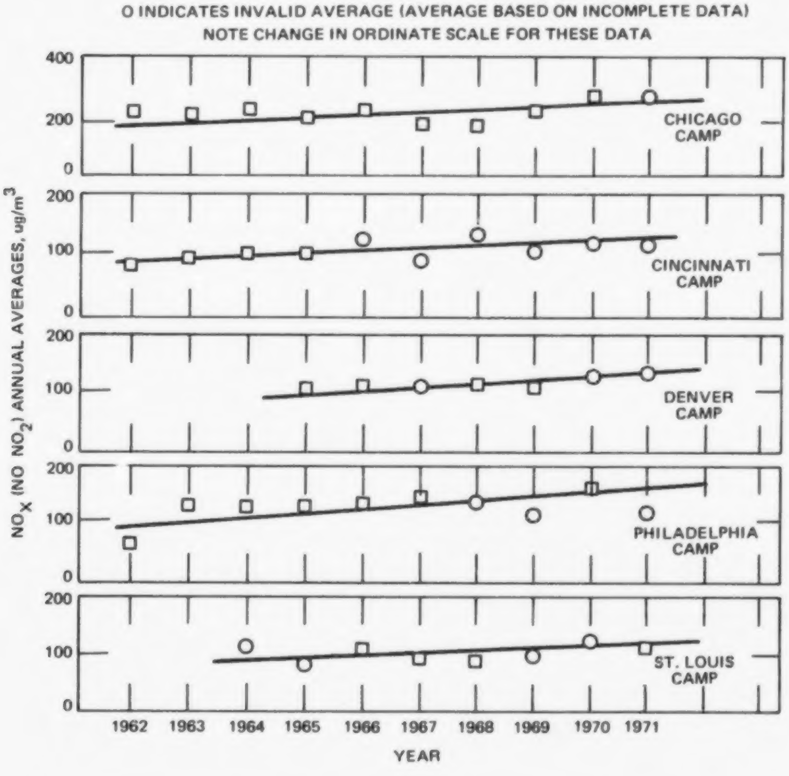

Figure 3-9. *Trend Lines for NO_x Annual Averages in Five CAMP Cities*

Source: *Air Quality and Emissions Trends Annual Report: Volume I.* Research Triangle Park, North Carolina: National Air Monitoring Program, Environmental Protection Agency, 4-28, 1973.

Symptomatology

There is no reliable "typical" symptom to aid the observer in diagnosing NO_2 injury. The extent and type of leaf injury or other effects can be affected by many factors. Short-term exposures to high NO_2 concentrations (*acute* exposures) do not necessarily result in the same symptoms as long-term exposures to lower concentrations (*chronic* exposures). The relative sensitivity of plants to NO_2 varies from species to species, and even from cultivar to cultivar within a species. Climate and other external factors can influence the expression of injury.

The one primary symptom of *chronic* NO_2 exposure is chlorosis, but since chlorosis *per se* is non-specific and can be a symptom of injury caused by other pollutants, it is therefore a very poor diagnostic tool for NO_2. Fortunately for field-assessment purposes, both chlorosis and necrosis often occur on the same plant when it is attacked by NO_2: older leaves tend to become chlorotic and younger leaves may show necrosis. Leaf chlorosis resulting from extended or recurrent exposures to relatively low NO_2 concentrations may be a transitory symptom and not a permanent one. After chronic NO_2 exposure is stopped, young leaves often recover and become green again, but chronic NO_2 exposure initiates irreversible aging in older leaves and the leaves often become dry and drop.

For many chronic exposures, chlorosis precedes the appearance of necrotic lesions. Cereal grains and corn leaves often develop longitudinal chlorotic bands before necrosis develops. In narrow-leaved monocots, chlorotic zones may occur as transition zones between healthy tissues and the necrotic tips. On some broad-leaved plants, chlorosis from chronic NO_2 exposures begins with many small yellow-green areas over the leaf surface which may merge as the exposure continues. In some species, chlorosis may be concentrated near the leaf margins.

There are no common plants that can serve as "bioindicators" for diagnosing NO_2 injury in the field.

Summarized below are general symptoms resulting from acute NO_2 exposures, and symptoms that may be induced by chronic NO_2 exposures. But even by using these descriptions as guides, NO_2 will still rarely be confirmed as being responsible for symptoms observed in the field. At best, it may only be possible to exclude NO_2 as the causal agent.

Broad-leaved (Dicotyledonous) Plants

Injury to leaves of broad-leaved plants that results from acute NO_2 exposure is usually characterized by the rapid appearance of irregularly-shaped intercostal lesions. The earliest indications of injury are gray-green watersoaked areas located on the upper leaf surface (Plate 3-1). Tissues in these areas collapse, become dry and bleached, turn white-to-tan, and extend through to the lower leaf surface (Plates 3-31, 3-32, 3-33). The resulting necrotic lesions are usually indistinguishable from lesions produced by sulfur dioxide.

Lesions on most broad-leaved plants are distributed between the veins throughout the leaf blade. NO_2-induced necrotic lesions may fall from the leaf, leaving irregular holes with darkened margins. Occasionally, the lesions may increase in size, coalesce, and form necrotic strips between the veins.

In some plants the injury tends to be more marginal. For example, necrosis on maple and oak leaves often begins at the margins or the tips of the lobes and extends into the mid-portions of leaves. For species with finely dissected compound leaves, such as carrot and parsley, NO_2 injury is usually confined to the tips and margins. Very high NO_2 exposures of citrus trees may result in defoliation of young leaves without the development of necrosis.

Narrow-leaved (Monocotyledonous) Plants

Acute NO_2 exposures of narrow-leaved plants most often result in yellow-to-ivory-to-white necrosis that begins at or just below the tips of leaf blades. Necrotic margins and striped necrotic lesions between the veins also occur. In most grains and grasses, injury from acute exposure affects the entire width of the leaf blade. The length of the affected portion varies with the NO_2 exposure. Grains also show longitudinal necrotic strips between the veins, and these can coalesce to form large necrotic areas on the leaf surface. The awns (beards) of rye and barley spikes are also susceptible to NO_2 injury. Bleached necrosis begins at the tips and progresses downward.

Coniferous Plants

Injury to leaves of conifers from acute NO_2 exposures usually begins at the tips of the needles and progresses towards the base. The boundary between healthy and injured tissues is sharply delineated by a brown or red-brown band. In the initial stages of injury, the tips of needles take on a dull, gray-green color which, in time, becomes light brown, then dark brown or red brown. Young emerging needles show NO_2 injury at the tips, whereas older needles may occasionally develop necrosis in the central or basal portions of needles.

Injured needles may drop prematurely; spruce needles drop shortly after injury develops; larch and fir needles may not fall for several months; and injured pine needles can remain on the tree for more than a year. However, if injury is severe, with necrosis covering more than half of the needle surface, defoliation usually occurs within a month.

Factors Affecting Plant Response

The extent, severity, and type of foliar lesions caused by a given NO_2 exposure can be affected by both external and internal factors. Climatic and soil conditions are the external factors, and the internal factors are plant genetic make-up, stage of development, age, etc.

External Factors

The effects of external factors such as light, temperature, relative humidity, and soil moisture on the sensitivity of plants to NO_2 are not well known. Night fumigations usually result in more severe injury than comparable daytime exposures; however, the progression of symptoms from incipient injury to dry necrotic lesions is most rapid on warm sunny days. Reduced soil moisture or low relative humidity make some plants more tolerant to NO_2.

The presence of other pollutants in the atmosphere with NO_2 adds another variable. When NO_2 and SO_2 occur together in the atmosphere, each at a concentration that is not harmful to plants, their combined effect can result in plant injury. A similar interaction has been reported for NO_2 and O_3. NO_2 may possibly interact in this manner with other air pollutants as well.

Internal Factors

Genetic effects are obvious when one compares the differences in sensitivity among species, cultivars, or clones of the same species. The age of leaves can affect their sensitivity to NO_2, as in tobacco, in which the oldest leaves turn yellow, the middle-aged leaves become yellow with necrotic lesions, and injury to the younger leaves is limited to necrosis. Middle-aged leaves are most sensitive in dandelion, cheeseweed, lamb's-quarters, pigweed, and Kentucky bluegrass, whereas the sensitivity of middle-aged and old leaves is about the same in sunflower, annual bluegrass, and nettle-leaf goosefoot. Emerging or elongating needles of conifers are more sensitive than mature needles.

Injury Threshold Doses

The effects of NO_2 on plants are not only dependent on the pollutant concentration, but are also influenced by the duration of exposure.

Threshold doses are described as functions of the pollutant concentration and exposure time, as shown in Figure 3-10.

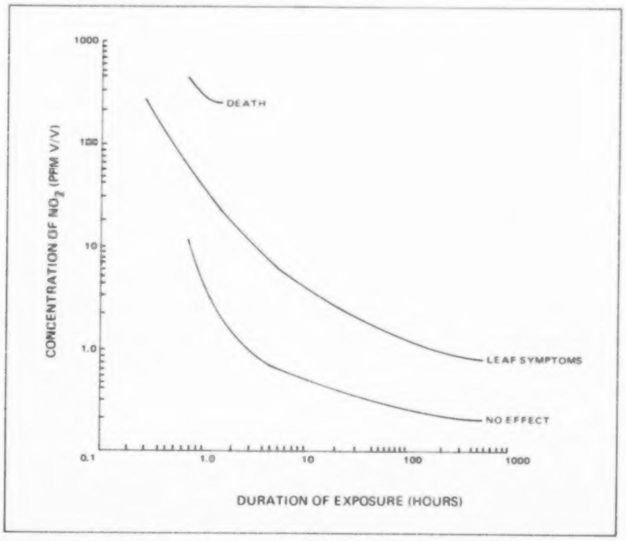

Figure 3-10. *Approximate Thresholds for Death, Leaf Injury, and No Effect, as Related to NO_2 Concentration and Duration of Exposure.*

For every effect of NO_2, a threshold curve can be computed if enough information is known about that effect over a relatively wide range of concentrations and exposure periods. In Figure 3-10, three threshold curves are shown. These are approximate, rather than absolute, and are based on information from experimental exposures of many plant species. The threshold for NO_2 doses that result in the death of plants is based on a limited amount of information; the threshold for leaf symptoms (chlorosis or necrosis) is based on many observations. In the area between the threshold curves for leaf symptoms and no effect are NO_2 doses that do not injure leaves, but result in reduced growth, or effects on photosynthesis, or effects on other plant processes.

Air Quality Standards

The current Federal Air Quality Standards for NO_2 are 0.05 ppm (94 $\mu g/m^3$) for an annual average, and 0.13 ppm (244 $\mu g/m^3$) for a 24-hour average. In addition to providing protection for most vegetation against the direct effects of NO_2, these standards may also limit the amount of NO_2 available for the production of photochemical oxidants, thereby providing some protection against the production of these pollutants as well.

Relative Sensitivity of Plants to NO_2

The relative sensitivities of some plant species to NO_2 are shown in Table 3-5. The three classes, sensitive, intermediate, and tolerant, should be considered approximate because they are based on subjective criteria from several sources. Most of the classifications are derived from experimental fumigations carried out at various locations at different times of the year, using different NO_2 exposures. Methods for assessing injury (e.g., percentage of leaves injured; amount of leaf surface affected; defoliation; etc.) were also variable. For these reasons, a plant species considered tolerant by one investigator may be considered sensitive by another.

Table 3-5

Relative Sensitivity of Various Plants to NO_2

	Conifers	
Sensitive	*Intermediate*	*Tolerant*
European larch	Spruce	Yew
Japanese larch	Fir	Pine
	Deciduous Trees and Shrubs	
Sensitive	*Intermediate*	*Tolerant*
European white birch	Norway maple	Black locust
	Japanese maple	European hornbeam
	Linden	Elder
		Gingko
		Elm
		Purple beech
		Oak

Table 3-5 (Continued)

Relative Sensitivity of Various Plants to NO_2

Fruit Trees

Sensitive	*Intermediate*	*Tolerant*
Apple	Orange	
Pear		

Field Crops and Grasses

Sensitive	*Intermediate*	*Tolerant*
Oats	Rye	Kentucky blue grass
Barley	Annual blue grass	
Red clover	Corn	
Spring clover	Wheat	
Spring vetch	Potato	
Alfalfa	Field beans	
Tobacco		

Garden Crops

Sensitive	*Intermediate*	*Tolerant*
Lettuce	Bush bean	Kohlrabi
Peas	Celery	Cabbage
Carrot	Tomato	Onion
Parsley		Asparagus
Leek		
Rhubarb		
Pinto bean		

Ornamental Shrubs and Flowers

Sensitive	*Intermediate*	*Tolerant*
Snapdragon	Brittlewood	Dahlia
Tuberous begonia	Pittosporum	Carissa
Rose	Ligustrum	Croton
Sweet pea	Ixora	Shore juniper
Oleander	Cape jasmine	Daisy
Bougainvillea	Gardenia	Lily of the Valley
Azalea	Petunia	Gladiolus
Hibiscus	Fuchsia	Plantain lily
Pyracantha	Catawba rhododendron	Heath

Weeds

Sensitive	*Intermediate*	*Tolerant*
Mustard	Dandelion	Nettle-leaf goosefoot
Sunflower	Chickweed	Lamb's-quarters
	Cheeseweed	Pigweed

Leaf Tissue Analysis

Chemical analyses of leaves, used to confirm suspected symptoms for some air pollutants, are of little value for NO_2 injury, due to the high and variable levels of nitrogen compounds that are normally found in plant tissues.

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Plate 3-1. *Example of Initial Water-Soaked Appearance on Broccoli Immediately after Exposure. This Plant was Fumigated with 10 pphm* O_3 *and 0.5 ppm* SO_2

Plate 3-2. *Bel W-3 Tobacco Injured by Ambient Oxidant in St. Louis, Mo. Showing Both Fleck and Bifacial Necrosis*

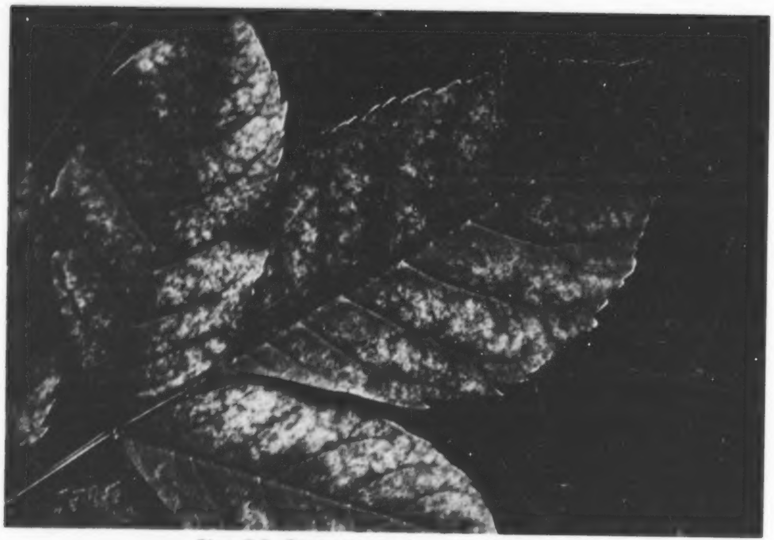

Plate 3-3. *Ozone Injury (Flecking) to White Ash*

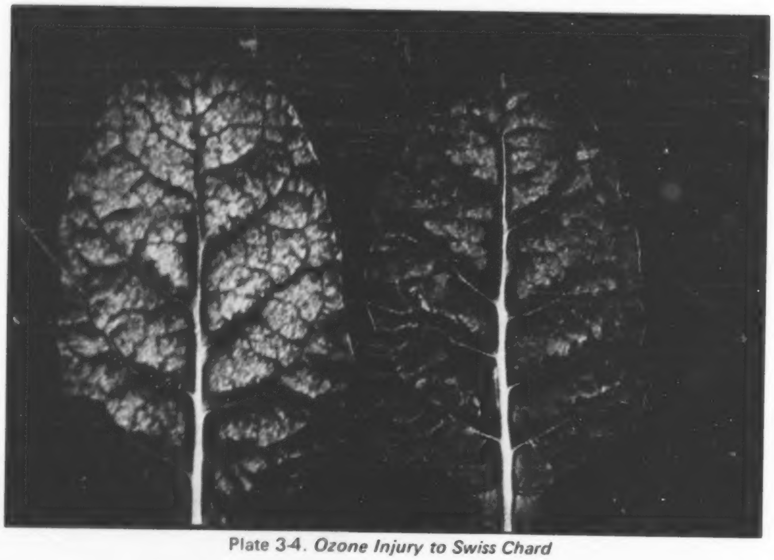

Plate 3-4. *Ozone Injury to Swiss Chard*

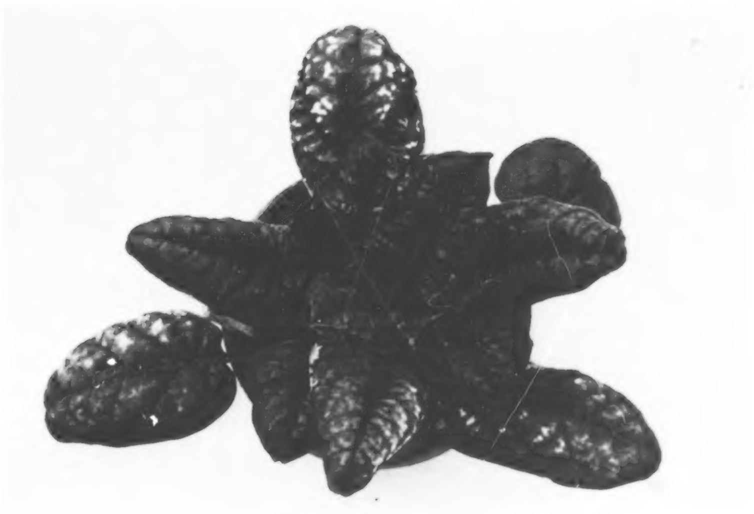

Plate 3-5. *Ozone Injury to Tobacco*

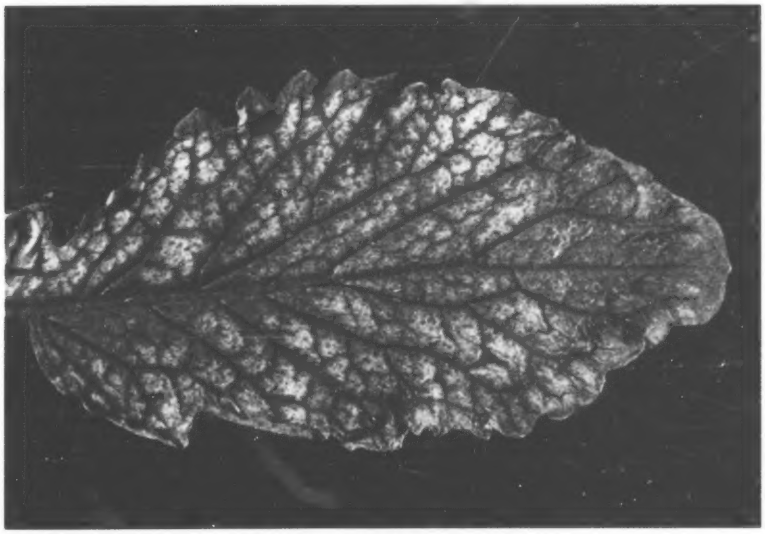

Plate 3-6. *Ozone Injury to Radish*

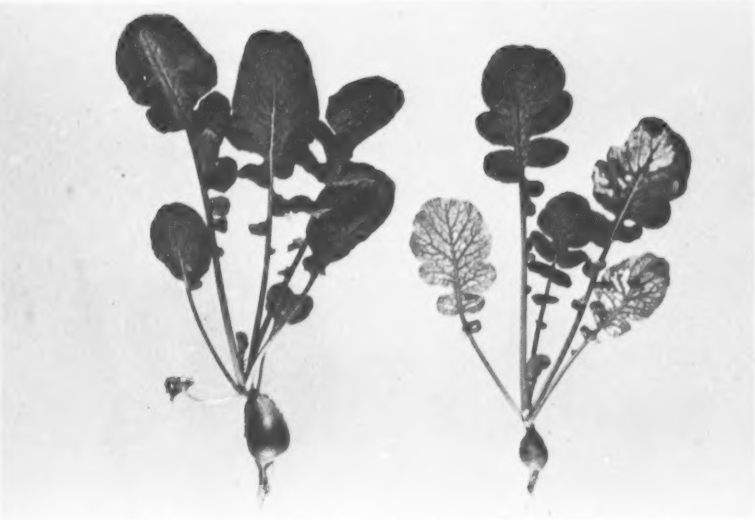

Plate 3-7. *Ozone Injury to Radish and Subsequent Reduced Yield on Right*

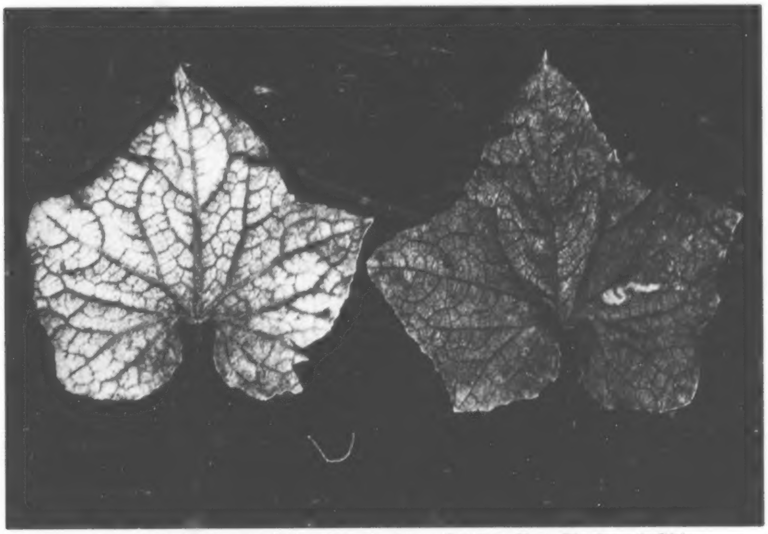

Plate 3-8. *Cucumber Injured by Ambient Oxidant Near Cincinnati, Ohio*

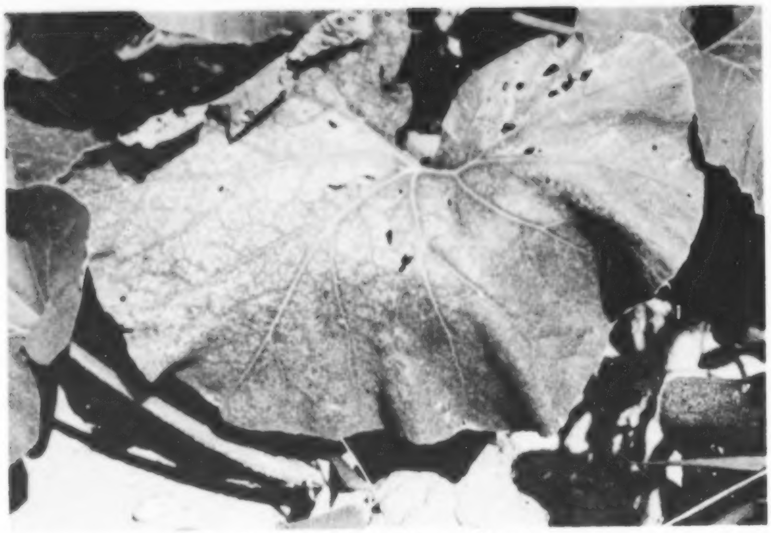

Plate 3-9. *Oxidant Injury to Squash*

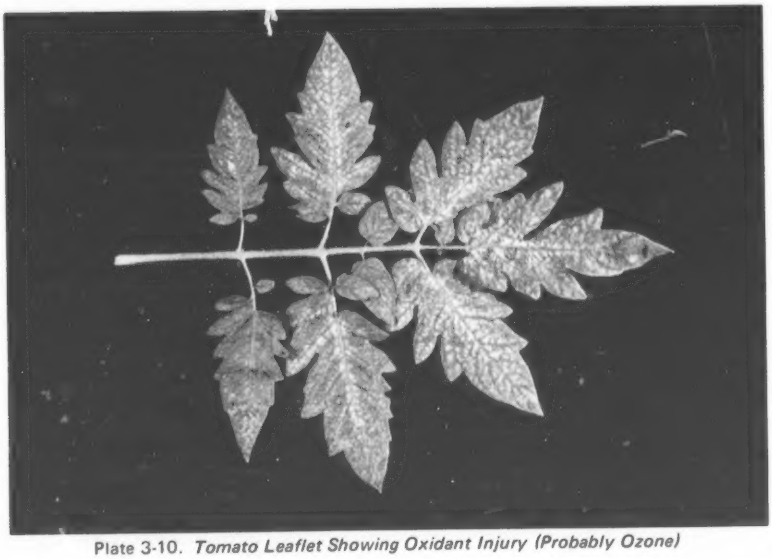

Plate 3-10. *Tomato Leaflet Showing Oxidant Injury (Probably Ozone)*

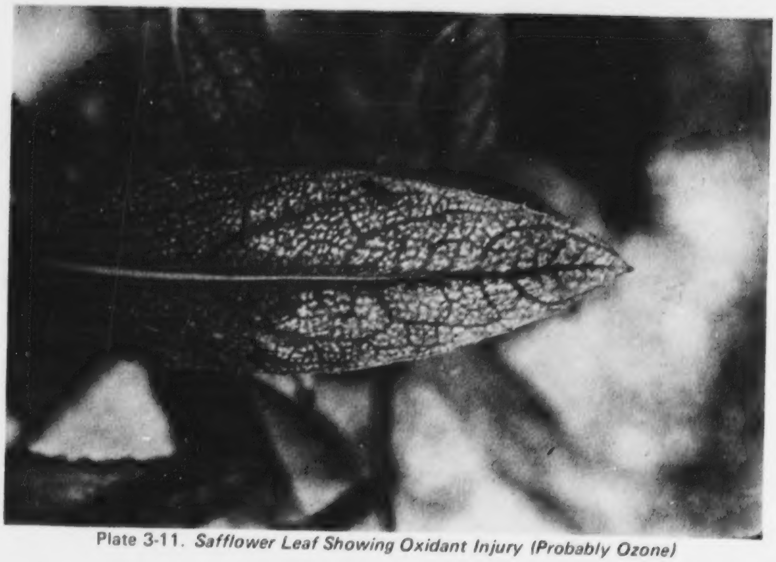

Plate 3-11. *Safflower Leaf Showing Oxidant Injury (Probably Ozone)*

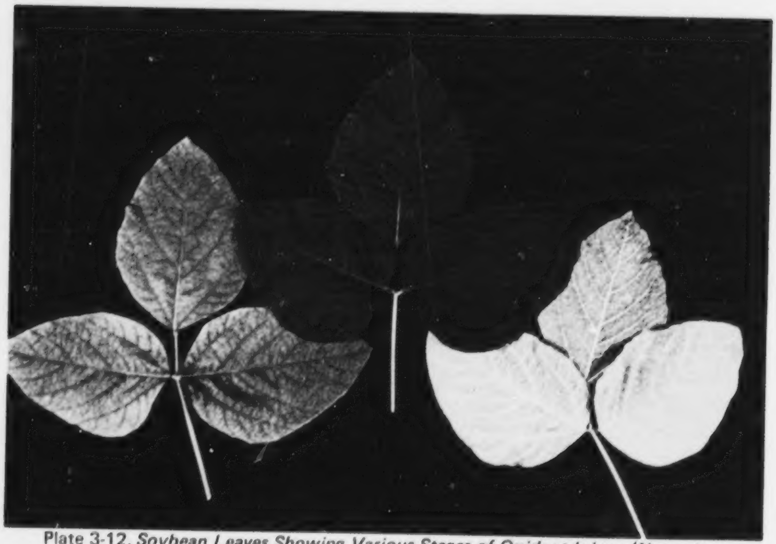

Plate 3-12. *Soybean Leaves Showing Various Stages of Oxidant Injury (Normal Leaf in Center; Leaflet on Right is Secondary Response to Oxidants)*

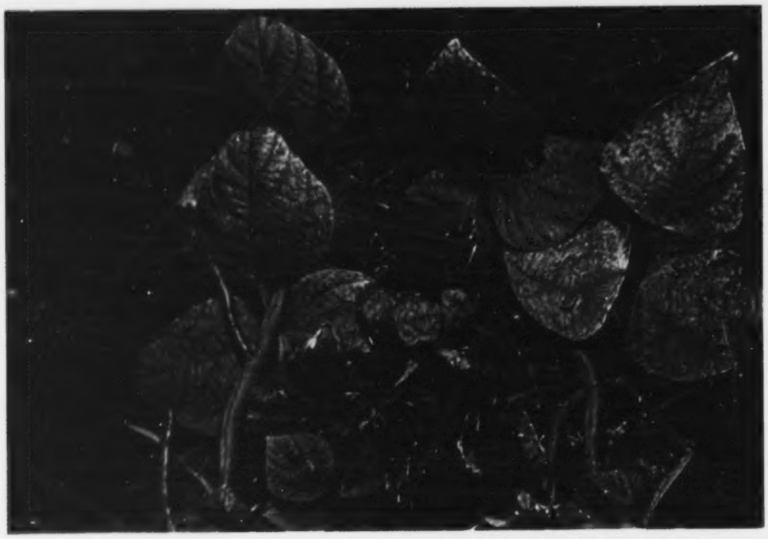

Plate 3-13. *Tempo Bean Leaves Showing Oxidant Injury (Probably Ozone)*

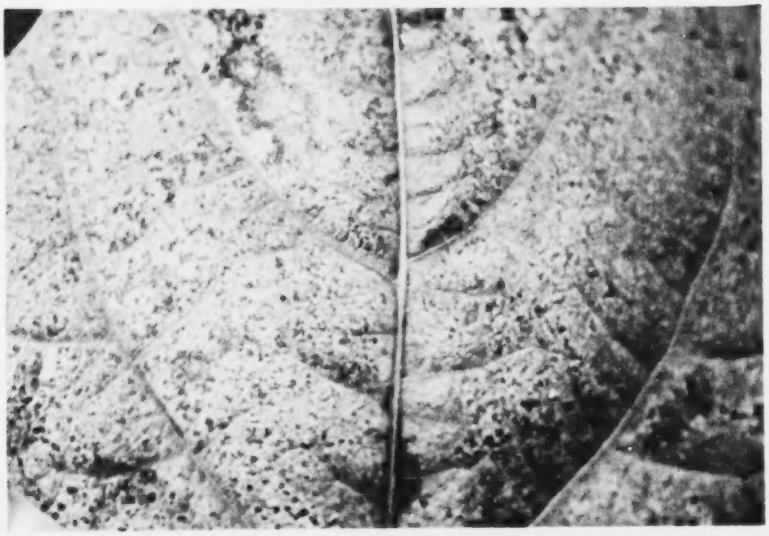

Plate 3-14. *Bronzing of White Bean Leaves Caused by Oxidant (Probably Ozone)*

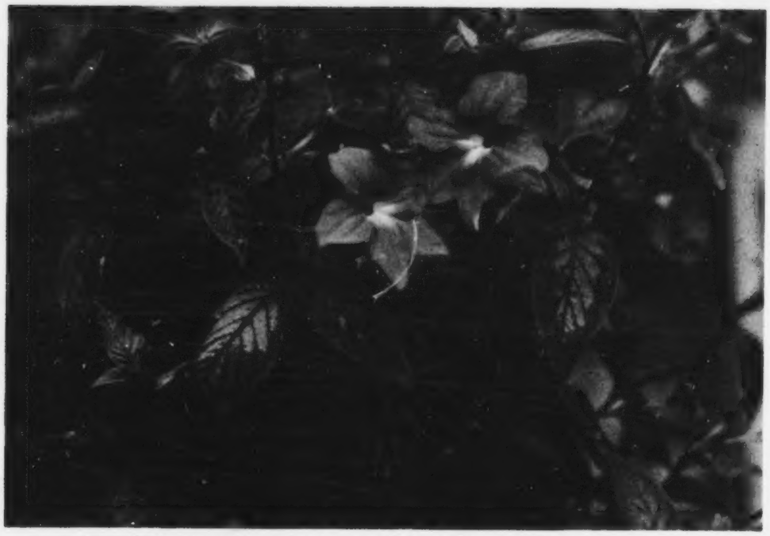

Plate 3-15. *Severe Ozone Injury and Bifacial Necrosis of Browallia Leaves*

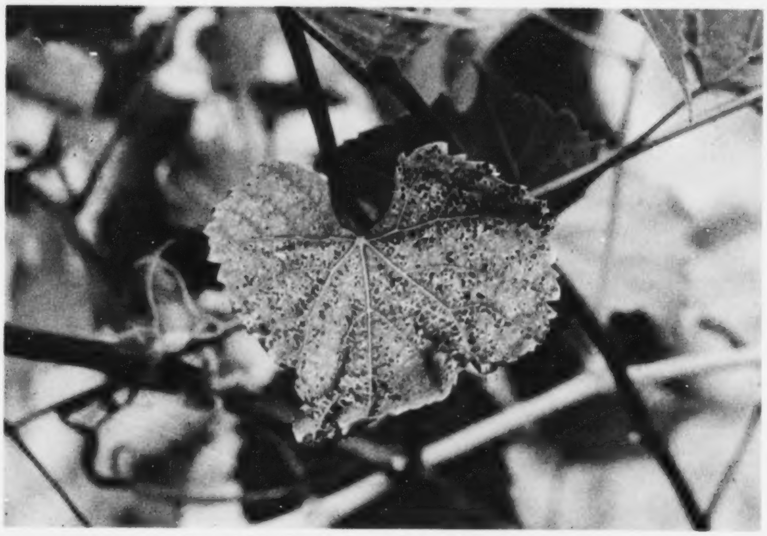

Plate 3-16. *Severe Grape Stipple Caused by Ozone*

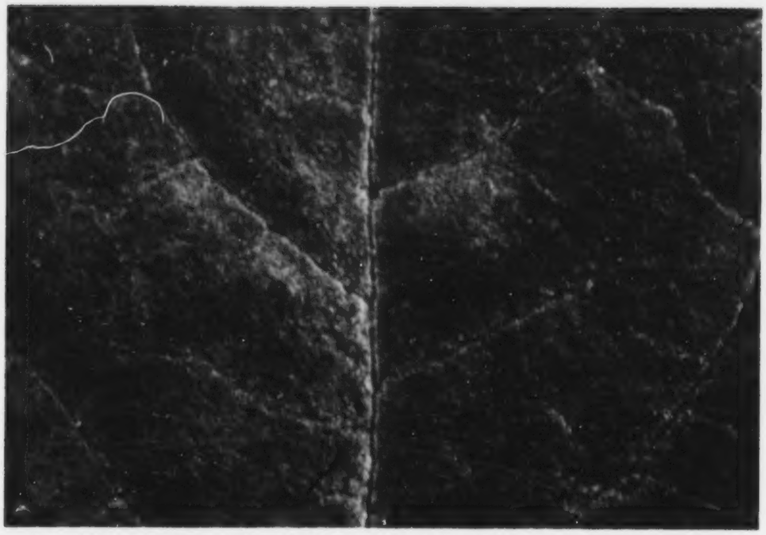

Plate 3-17. *Pinto Bean Injured by Ambient Oxidant Near Cincinnati, Ohio, Showing Stipple Symptom*

Plate 3-18. *Ozone Stipple of White Ash as Observed in the Field*

Plate 3-19. *Bluegrass Injured by Ambient Oxidant in the Los Angeles Basin*

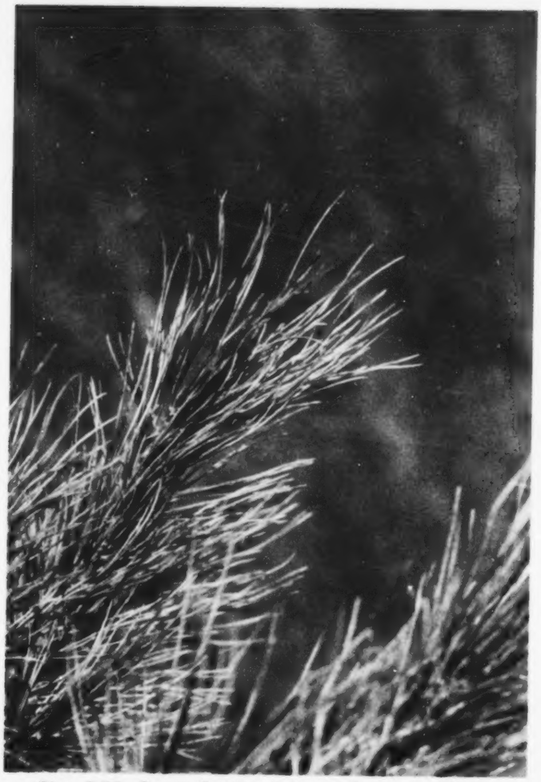

Plate 3-20. *Severe Oxidant Injury to Ponderosa Pine*

Plate 3-21. *Chronic Ozone Injury of Ponderosa Pine. Note Difference in Tolerance Between Trees and Tufted Appearance of Foliage*

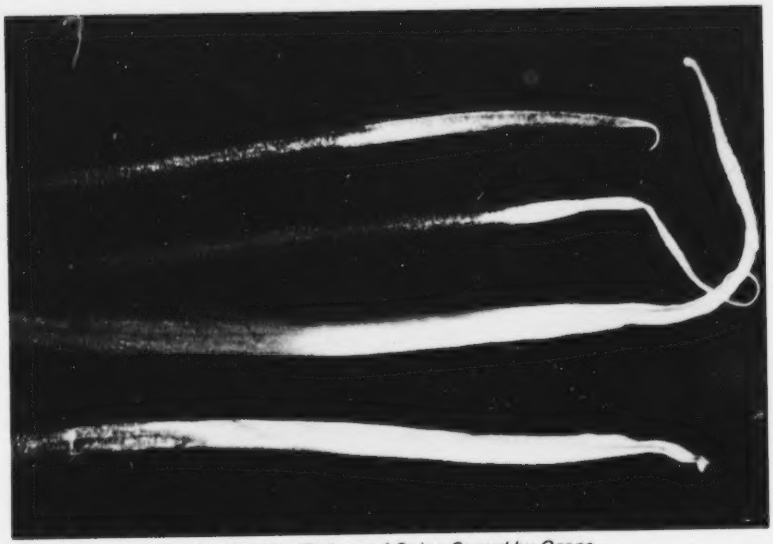

Plate 3-22. *Tipburn of Onion Caused by Ozone*

Plate 3-23. *Eastern White Pine Displaying Oxidant Injury (Probably Ozone)*

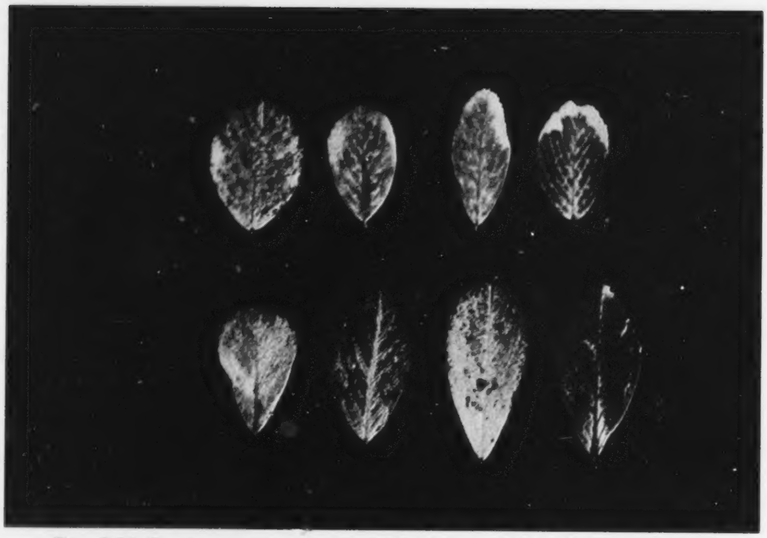

Plate 3-24. *Comparison of Ozone and PAN Injury on Alfalfa. Upper row: Ozone Injury; Lower row: PAN Injury*

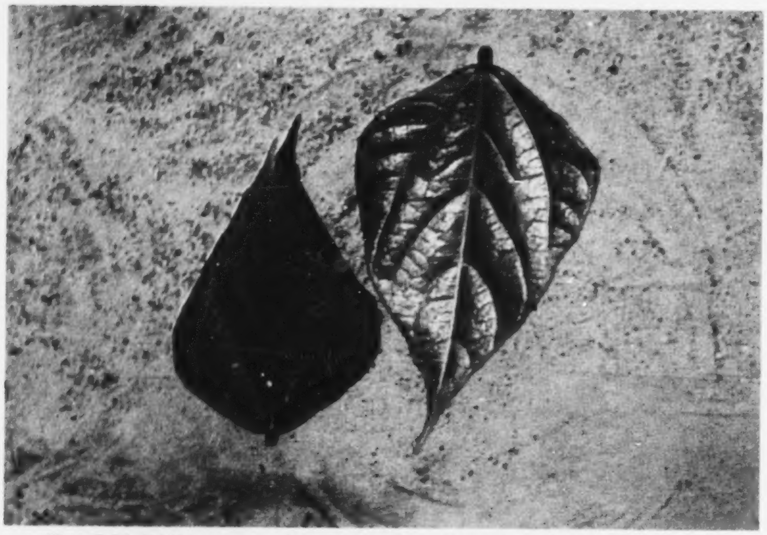

Plate 3-25. *PAN Injury to a Bean Leaflet. Left, Upper Leaf Surface; Right, Lower Leaf Surface Showing Silvering or Glazing Due to PAN*

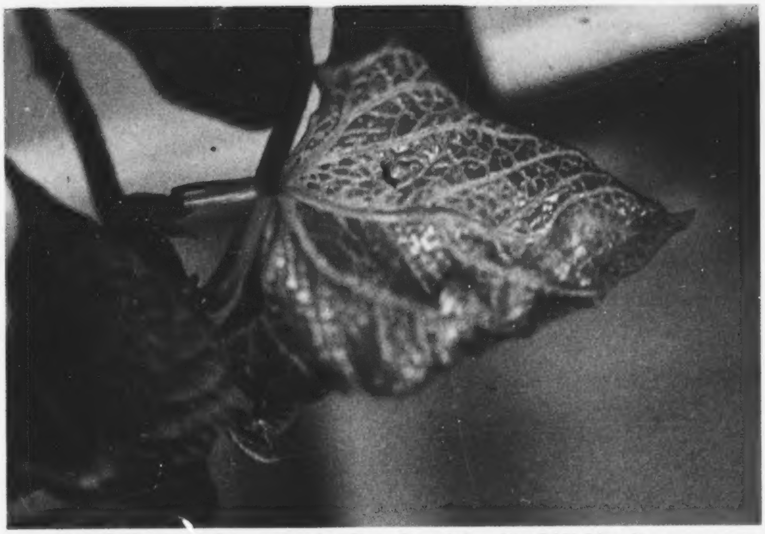

Plate 3-26. *Young Primary Leaf of Pinto Bean Injured by PAN After Exposure to Irradiated Auto Exhaust*

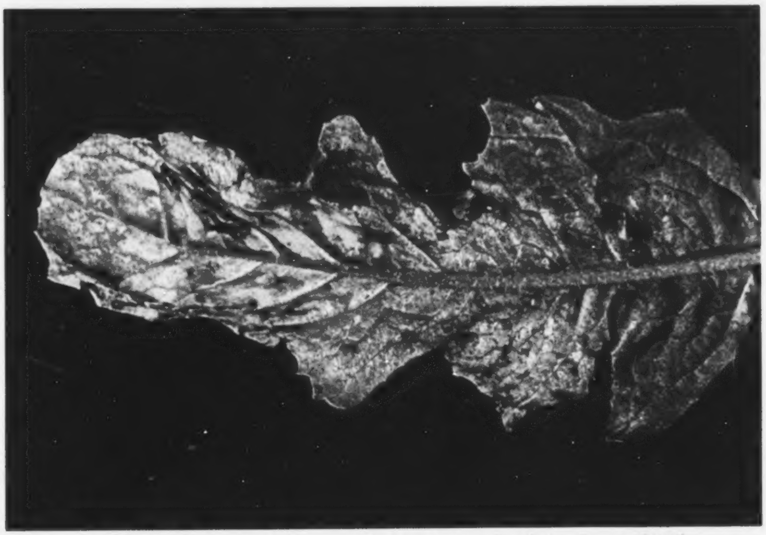

Plate 3-27. *Escarole Showing Oxidant Injury (Probably Due to PAN)*

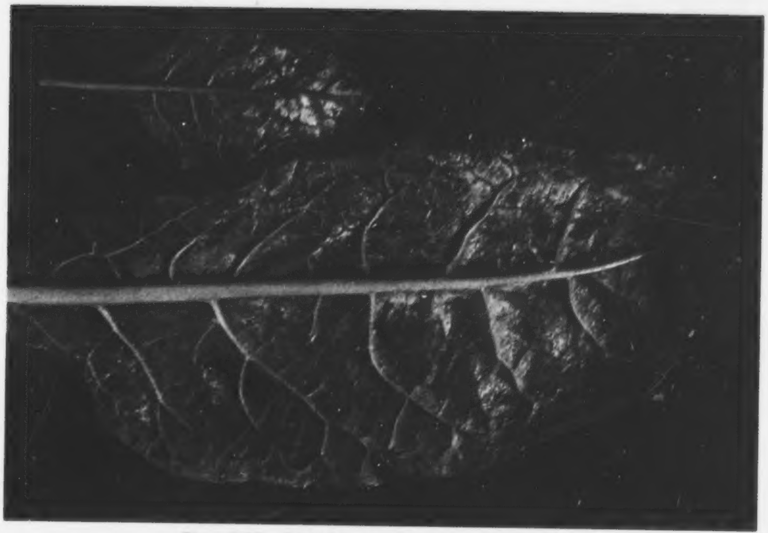

Plate 3-28. *PAN (Ambient) Injury to Swiss Chard*

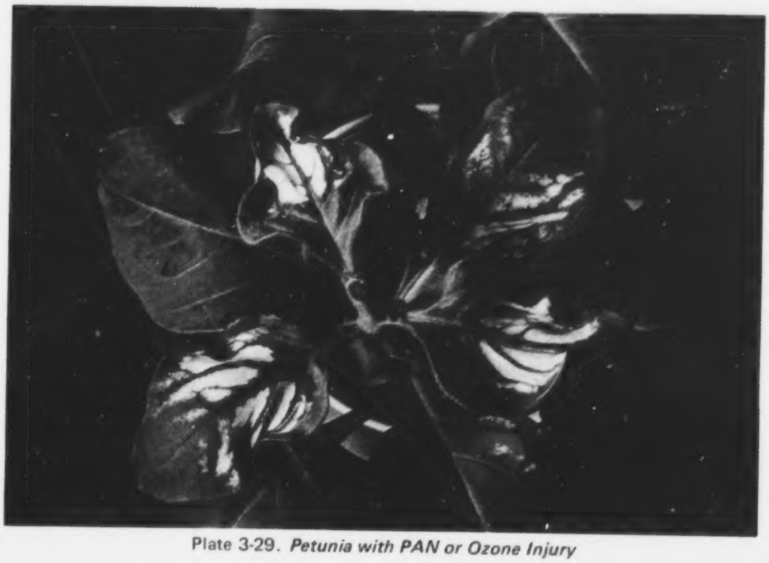

Plate 3-29. *Petunia with PAN or Ozone Injury*

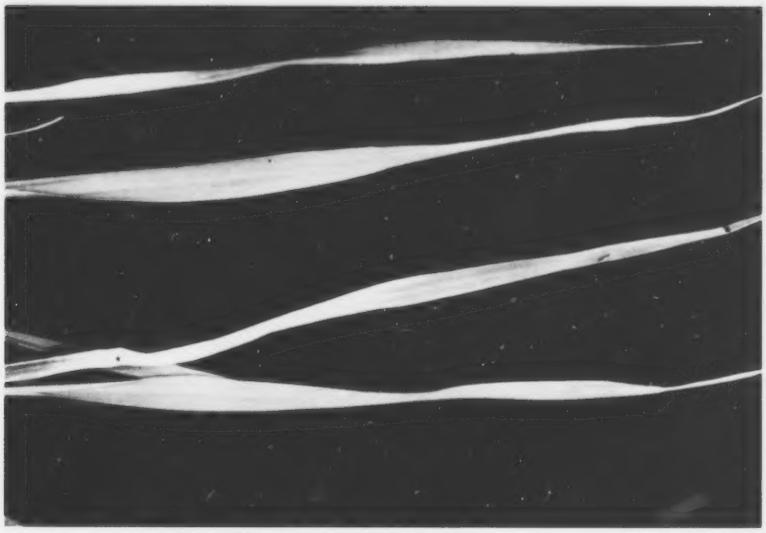

Plate 3-30. *PAN Injury to Oats*

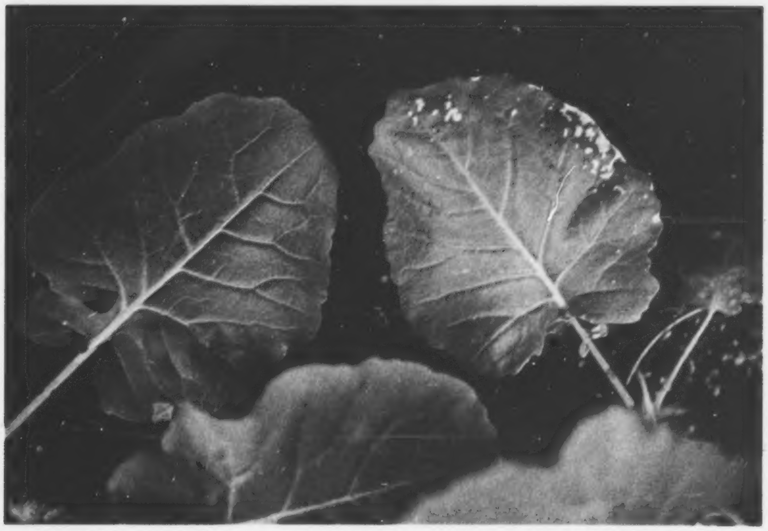

Plate 3-31. *Injury on Broccoli One Week After Exposure to NO_2. Leaves Show Some Recovery from Initial Water-Soaking and Also Some Marginal and Interveinal Necrosis*

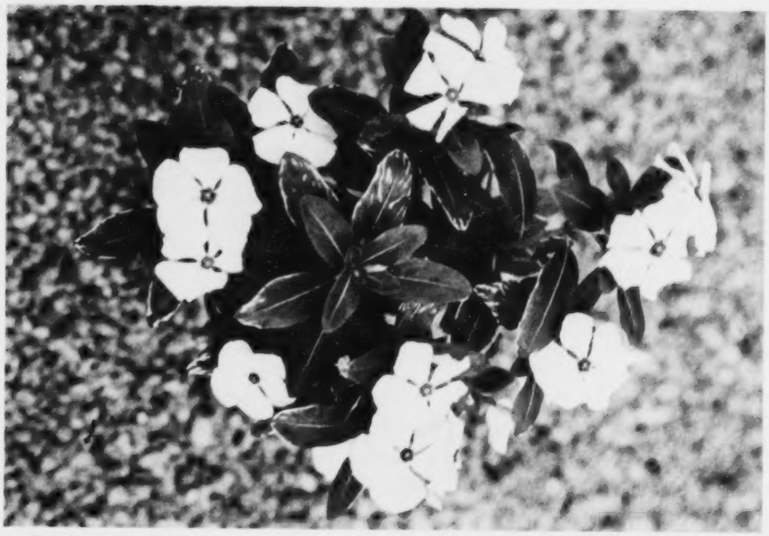

Plate 3-32. *Marginal and Interveinal Injury on Periwinkle Fumigated with 19.3 ppm of NO_2 for 0.5 Hours*

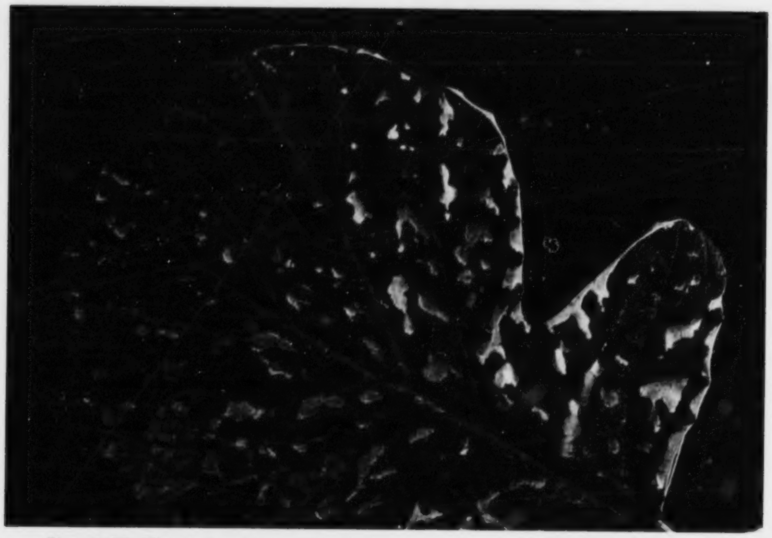

Plate 3-33. *Marginal and Interveinal Injury on Cherry Belle Radish Fumigated with 7.7 ppm NO_2 for Four Hours*

Chapter 4

SULFUR DIOXIDE

Introduction

Sulfur dioxide (SO_2) was one of the first man-made pollutants to cause economic damage to vegetation. Coming as it does from combustion of fossil fuels, plant materials, and refuse, green plants must have recorded its presence since early in the industrial revolution. Since that time, as industries and cities have expanded, concentrations of combustion products have continually increased.

Sources

In addition to combustion, sulfur dioxide is released when ores containing sulfur are roasted in the smelting process, when oils are refined, and when sulfuric acid is manufactured. In all these processes, the sulfur present in the materials is released by heat, whereupon it reacts with oxygen in the air to form SO_2.

The size (volume) of the emitters and their height and numbers are of considerable importance in influencing ground-level SO_2 concentrations. The trend in industrial operations has been away from low-level sources and toward releasing large volumes of pollutants from tall stacks (some reaching 600 to 1,200 feet high). Such high-elevation emission points disperse the pollutants over a wide area, with dilution normally adequate to avoid any effect on vegetation at ground level.

With the development of tall stacks in this century, a significant change has occurred in the "ground pattern" of SO_2 plant injury. During the early part of the century, plant damage frequently occurred in rather well-defined areas around a particular industrial operation, with the damage being most severe nearby the source. In those days, SO_2 acquired the name "smelter smoke," and plant damage became known as "smoke injury." However, with the use of tall stacks and controls on large SO_2 emitters, the occurrence of severe plant damage to many species in restricted areas has all but disappeared.

During the 1950's and 1960's, widespread damage from SO_2 occurred to highly sensitive plant species in the New York megalopolis, and in other cities where coal or oil was burned. Injury on tulip, violet, and crabapple was readily observed in such areas. The development of injury on so few plant species over a rather large area, with no apparent centers of increased injury, suggested that relatively smaller quantities of the phytotoxicant were coming from many widespread, rather small emitters, perhaps space heaters.

Atmospheric Chemistry

Laboratory and field studies show that SO_2 may be oxidized to SO_3 under conditions occurring in ambient air. This trioxide rapidly combines with water to form sulfuric acid mist. The mist consists of small airborne droplets of sulfuric acid (H_2SO_4), which vary in size with the relative humidity. Humidity, sunlight, and catalytic particulate matter (such as manganic and manganous salts, aluminum oxide, active carbon, and even coal soot) all affect the production of acid particles.

In addition, when photochemical-smog reactions involving unsaturated hydrocarbons and nitrogen oxides occur in the presence of SO_2, oxidation of the SO_2 to H_2SO_4 occurs at a significant rate. This happens even with concentrations of hydrocarbons and nitrogen oxides that are typical of the levels occurring in only moderately-polluted atmospheres (about 0.1 ppm). A similar oxidation occurs in the presence of ozone-olefin reactions, which may take place in the dark.

The rate of SO_2 oxidation to H_2SO_4 reportedly ranges from 0.1% to 10% per hour. Reactions between SO_2 and ammonia in the atmosphere also occur rapidly, to form ammonium sulfate. Droplets of sulfuric acid and particles of ammonium sulfate tend to settle in time but reduce visibility while suspended. In addition, SO_2 is readily dissolved in rain or snow, and settles to the earth's surface in those forms. This solubility in water suggests that the oceans may be a major sink for atmospheric SO_2. In addition, plant foliage and moist soil also serve effectively to remove this phytotoxic gas from the atmosphere.

Monitoring Data

To understand the problem of SO_2 pollution, it is helpful to know the quantities and sources of this phytotoxic pollutant, and the trends in total quantities and ground-level concentrations. Table 4-1 shows the estimated total SO_2 emissions in the U.S., 1940-1970. Table 4-2 shows the sources of SO_2 emissions for 1970.

Table 4-1

Estimated Total Nationwide SO_2 Emissions
Levels, 1940-1970

Year	*SO_2 emitted (10^6 tons/year)*
1940	22.8
1950	24.9
1960	23.2
1968	31.1
1970	33.4

Source:

Air Quality and Emissions Trends Annual Report: Volume I. Research Triangle Park, North Carolina: National Air Monitoring Program, Environmental Protection Agency, August 1973. p. 1-13.

Table 4-2

Nationwide Estimates of SO_2 Emissions from Various Sources During 1970

Source	*SO_2 in 10^6 tons/year*
Transportation	1.0
Fuel combustion in stationary sources	26.4
Industrial processes*	6.4
Solid waste disposal	0.1
Miscellaneous	0.2
Total	34.1

*Includes refinery operations, ore smelting, coke processing, and sulfuric acid manufacture.

Source:
Air Quality and Emissions Trends Annual Report: Volume I. Research Triangle Park, North Carolina: National Air Monitoring Program, Environmental Protection Agency, August 1973. p. 1-7.

Air-sampling data secured by the National Aerometric Surveillance Network show that in all four air quality Control Regions (Northeast, North Central, South, and West), the composite annual averages for both the arithmetic mean and the average maximum daily SO_2 concentrations exhibit a *downward* trend for the eight-year period 1964-1971 as summarized in Table 4-3.

Table 4-3

Sulfur Dioxide Annual Average Concentration of Four Air Quality Control Regions for 1964 and 1971, in $\mu g/m^3$

	YEAR	
Region	*1964*	*1971*
Northeast	88	41
North Central	49	24
South	34	14
West	25	14

Only one sampling site out of 32 showed a significant upward trend. During the last four years of the period only three out of 95 sampling sites revealed an upward trend. Thus, the SO_2 analysis work shows a pronounced *downward* trend, with the composite average dropping over 50 percent. However, a review of the data for the same period recorded in Table 4-1 shows a substantial *increase* in SO_2 emissions. How can this apparent inconsistency be accounted for? The following considerations offered by the National Air Monitoring Program help explain:

> First, emissions are determined for the nation as a whole, whereas air-quality data are generally collected from specific sites in center-city locations. Second, SO_2 emission rates in most urban areas are

declining. The use of coal in residential and small commercial sources is practically non-existent. Cleaner fuels such as natural gas and distillate fuel oils have replaced coal to a large extent. The impact on total nationwide emissions as a result of this fuel replacement is relatively small, but the effect on local air quality is pronounced. Third, large point sources such as power plants are not able to locate near or in center-city areas. Increased fuel transportation costs favor the generation of electricity near the fuel source.*

Symptomatology

It is universally accepted that SO_2 enters the leaves of plants through open stomata. Inside the leaf, large surface areas of moist cells that are oxygen-rich during daylight hours are exposed to this gas. In such an environment, SO_2 is oxidized to highly toxic sulfite (SO_3) and then to less-toxic sulfate (SO_4). When sublethal concentrations of SO_2 are absorbed by the cells, they oxidize to highly toxic SO_3, which is slowly oxidized to less-toxic SO_4 and neutralized in the plant cells. This reduces the toxicity of the sulfur by a factor of about 30.

If SO_2 is not absorbed too rapidly, a concentration of SO_4 may accumulate in the cells that is several times greater than that occurring in unexposed plants of the same species. Thus, plants exposed to sublethal concentrations of SO_2 may in time develop chronic sulfate (SO_4) injury. However, since the highly toxic SO_3 is slowly oxidized in the plant cells to the less-toxic sulfate form, it is apparent that, if SO_2 is absorbed more rapidly than SO_4 is formed, the highly phytotoxic SO_3 will accumulate in toxic proportions, causing acute injury.

Since the earliest investigations, alfalfa has been considered one of the most SO_2-sensitive plants. The acute injury symptoms to this plant usually consist of a bleaching appearance, and can occur both between the veins and marginally. The most typical symptoms are marginal, with the bleached areas extending progressively toward the midrib with increasing severity of fumigation.

Bracken fern (Plate 4-1) in woodlands is an extremely useful indicator plant. The injury is typically orange-red and marginal on the fronds. Wild and cultivated mustard and swiss chard are also excellent indicator plants. Their injuries are typically ivory-white and occur between the veins. The younger, expanding, and the older leaves remain free of injury. Injury to sow thistle is marginal and intercostal, and varies in color from gray to dark. In wooded areas, fox and bank grapes (Plate 4-2) are quite sensitive, and exhibit typical SO_2 intercostal and interveinal markings that are light brown.

White birch (Plate 4-3) and trembling aspen (Plate 4-4) are the most SO_2-sensitive deciduous trees. The markings on trembling aspen leaves are usually intercostal but often also occur on the margins. The necrotic areas are reddish-brown at first, but darken with age. A dark-colored line often develops between the injured and green tissue.

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*From: *Air Quality and Emissions Trends Annual Report: Volume I.* Research Triangle Park, North Carolina: National Air Monitoring Program, Environmental Protection Agency, August 1973.

In the South, the Brazilian pepper bush is an excellent SO_2 indicator. The spotting occurs interveinally on all but the youngest leaves. The color of the injury is a grayish-tan.

White pine and jack pine are the most sensitive conifers. Since needles that are nearly mature or have recently reached full elongation are the most sensitive, conifers are especially sensitive during late spring and early summer. Needles showing acute injury develop an orange-red color. (An entirely green needle is rarely found in a sulfur dioxide-affected fascicle.)

Some SO_2 symptoms are highly similar to symptoms of other plant problems. Needle blight resulting from fungus infections often has green and blighted needles together in a fascicle. Diseased needles may show small, dark, spore-bearing fungus structures in an area at the base of the injured tissue. This is not visible to the naked eye but is clearly visible using a hand lens. The diseased area is usually a slightly darker brown than the necrotic terminal. On conifers, needle blight resulting from acute SO_2 toxicity might also be confused with injury caused by other atmospheric pollutants, such as chlorine, fluoride, and even cold temperatures.

Broad-leaved (Dicotyledonous) Plants

Acute Injury. Acute injury due to rapid absorption of a toxic dose of SO_2 causes marginal and intercostal necrotic areas which at first have a dull, dark-green, watersoaked appearance. On drying and bleaching, these areas become ivory-to-white in most plant species, such as garden pea (Plate 4-5), corn (Plate 4-6), and alfalfa (Plates 4-7, 4-8). But in some species various shades of brown predominate. Examples include chrysanthemum, lettuce, blackberry (Plate 4-9), fox grape (Plate 4-2), hickory (Plate 4-10), and eggplant (Plate 4-11). In a few species, red predominates (e.g., quince, Plate 4-12).

The necrotic areas extend through the leaf and are visible on both surfaces. The areas immediately bordering the veins are seldom injured. These areas generally have few stomata and very limited intercellular spaces. The leaves most sensitive to SO_2 are younger, fully-expanded leaves, and those that are near full enlargement (Plate 4-13). Older leaves are less sensitive. The smaller, enlarging leaves are the last to show acute injury.

Histological examination of a lesion that is beginning to develop reveals collapse of mesophyll cells near the stomata. Mesophyll cells collapse not only from SO_2 toxicity, but from other causes as well. The absence of collapsed mesophyll adjacent to necrotic areas can definitely eliminate SO_2 as a cause of necrosis, but the collapse of such tissue, because of its ubiquitous nature, does not definitely establish SO_2 toxicity as the cause.

There may be considerable variability in sensitivity to a phytotoxic pollutant among individual plants of a given species.

Chronic or Sulfate Injury. Plants exposed to sublethal concentrations of SO_2 may in time develop chlorotic symptoms (Plate 4-14), especially if the exposure is continued. (The symptoms may also be white or reddish-brown.) Such yellowing affects the margins and interveinal areas, with the affected areas remaining turgid. The loss of green color results from the plasmolysis of the chloroplasts in affected mesophyll cells, with the destruction of chlorophyll causing a bleached appearance. The veins on such leaves usually remain green.

In cotton, a yellowing first appears on the undersurface of the leaf which gradually extends through to the upper surface. This has been called "sulfate toxicity," and it frequently appears as white or brownish-red turgid areas between the veins. Such leaves usually drop prematurely. Silvering of the lower leaf surface may occasionally occur in cotton when gas concentrations and humidity are low. This response results from a collapse of the cells immediately internal to the epidermal cells.

A similar type of marking may occur following an acute SO_2 fumigation. The mild chlorosis results from accumulation of SO_3 at concentrations sufficient to bleach chlorophyll, but not sufficient to kill the cells. Evidence of chronic injury may occur on leaves that are also exhibiting acute symptoms described above.

Narrow-leaved (Monocotyledonous) Plants

Acute Injury. Monocot leaves vary in their response to SO_2 from injured tips at low concentrations, to almost completely necrotic leaves at high concentrations. Injury usually develops first at the leaf tips, then extends downward with increasing concentrations and exposure time. The markings that appear on grain crops, lilies, and gladioli usually appear as necrotic streaks between the veins, and usually appear first at the bend of the leaf when present (Plate 4-15).

Coniferous Plants

Acute Injury. In the case of conifers exposed to large dosages, needles develop a watersoaked appearance that soon changes to reddish-brown as shown in Plates 4-16 and 4-17. As lesions develop in white pine needles, they fill with resin. Fully-expanded needles, and those that are nearly so, exhibit the most necrosis. Very young needles are seldom injured, perhaps because of inactivity of the stomata in needles of that age. Conifer needles absorb more SO_2 and are much more sensitive to the gas during spring and summer months than during the autumn and winter period.

Chronic or Sulfate Injury. This is expressed by chlorosis, especially in the older needles. Such needles are underproductive and are dropped prematurely.

Factors Affecting Plant Response

Light

Researchers generally agree that stomata are the sole or principal avenue of ingress of gases into the leaf. The view has been expressed that SO_2 is not dependent upon stomata for entrance into the leaf, but common experience contradicts this: plants that close their stomata at night become highly tolerant during this period, while plants whose stomata remain open at night (such as the potato) retain SO_2 sensitivity during the dark hours. In fact, potato foliage is *more* sensitive to SO_2 during dark hours than during the daylight. Sensitivity is also considerably greater in the morning than in the late afternoon.

Soil Moisture

The degree of turgidity of leaves at the time of fumigation is one of the most significant factors in SO_2 sensitivity. This has been shown by fumigation studies of turgid and wilted foliage from four species of plants. In these experiments, SO_2 concentrations ranged from 0.82 ppm (2,148 $\mu g/m^3$) to 4.0 ppm (10,480 $\mu g/m^3$). Injury on turgid plants ranged from moderate to very severe (involving all leaves), while the wilted foliage exhibited little or no injury, even at the highest concentrations used. The pronounced difference is believed to be due, at least in part, to the condition of the stomata, which close during times of moisture stress.

Many investigations have reported great increases in tolerance of plants to SO_2 as soil moisture approaches the wilting point. On the other hand, when soil moisture is adequate for growth, wide variations in moisture do not influence sensitivity in barley and alfalfa plants. Thus, it is believed that soil moisture influences sensitivity mainly through its effect on the stomata. When the soil moisture is low the stomata tend to close, thus limiting the ingress of SO_2.

Humidity

Humidity also exerts a marked influence on stomatal openings and plant response to SO_2. Plants at 30% relative humidity are about three times as tolerant to SO_2 than plants at 100% relative humidity. However, variations in the range between 50% and 75% relative humidity do not significantly affect plant sensitivity. The sensitivity of alfalfa to SO_2, for example, is not influenced to a great extent by changes in relative humidity between 40% and 70%.

Temperature

The toxic effect of SO_2 is at a minimum during exposures at low temperatures. For example, conifers during winter months are more tolerant to SO_2. Toxic effects increase with rising temperatures, within certain limits. There appears to be a critical temperature of 7°C, above which plant injury may occur in nature. In addition, several investigators consider that plant response to SO_2 is not markedly affected between 18°C and 40°C, providing other influencing factors are uniform.

Nutrition

From field experience it has been repeatedly observed that trees, vegetables, and flower crop plants that are growing well are more responsive to SO_2 exposures than are plants of the same species that are not growing well. This observation was confirmed by researchers when they fumigated plants deficient in nitrogen or sulfur. The plants exhibited reduced susceptibility and increased stomatal resistance to SO_2, as compared with plants that were making better growth at higher nutritional levels.

Also, tobacco and tomato plants grown at various sulfate levels in nutrient solution and exposed to SO_2 fumigations showed increased sensitivity to SO_2 with increased sulfate nutrition. The increased injury paralleled elevated foliar-total sulfur absorption from both the nutrient solution and atmospheric SO_2.

However, it has also been shown that absorption of $S^{35}O_2$ from SO_2 fumigations was least by plants growing in nutrient solutions high in sulfur content. Still other researchers have found that varying the sulfur content of the nutrient supply of alfalfa and buckwheat had no significant effect on the sensitivity of these plants to SO_2.

Sulfur dioxide absorbed from the atmosphere has been found to be present in greater concentrations in the inorganic fraction of test plants, rather than in the organic. However, when sulfur-deficient tomatoes were exposed to $S^{35}O_2$, about one-half of the absorbed sulfur had been converted into organic forms within six hours, thus demonstrating the nutrient value of SO_2.

Sulfur absorbed from the atmosphere has also been found in reduced concentrations in the roots. Sulfate formed in the leaves of alfalfa is held quite tenaciously; however, a considerable amount of sulfate formed in leaves of tomatoes is translocated. It is of interest also that a considerable concentration of sulfate has been found in the soil around the roots of treated plants, suggesting a passage outward of the sulfate from the roots. It has been suggested that atmospheric SO_2, absorbed by the leaves, moves inside the plant and can be eliminated through the roots.

Plant and Tissue Age

Full-grown and nearly full-grown leaves are among the most sensitive to SO_2. Young and old leaves usually exhibit less sensitivity to this pollutant (Plate 4-13). For example, uptake of gaseous SO_2 by leaves of Bel W-3 tobacco plants is high in the intermediate age and low in young and old leaves. It is suggested that these differences in SO_2 uptake could account for the visible injury patterns observed on plants that have been exposed to a phytotoxic concentration of SO_2. In addition, because plants that are growing well are more sensitive than those that are nutrient-deficient, it may be possible that physiological activity of the leaf contributes to its own sensitivity.

Injury Threshold Doses

O'Gara, in an attempt to classify plants on the basis of their sensitivity, fumigated about 300 species and cultivars of plants. He used alfalfa to represent the most sensitive of plants, giving it a rating of 1. He found that injury could be induced on this plant when it was exposed to a concentration of 1.2 ppm (3,144 $\mu g/m^3$) SO_2 for one hour. The rating he gave to a plant was determined by taking the concentration of SO_2 required to induce injury during a one-hour exposure and dividing it by 1.2.

Canadian studies in an area containing smelters near Sudbury, Ontario, classified a plant's sensitivity to SO_2 as 100 when it responded to an average concentration of SO_2 of 0.95 ppm (2,489 $\mu g/m^3$) for 1 hour, or 0.55 ppm (1,441 $\mu g/m^3$) for two hours, or 0.35 ppm (917 $\mu g/m^3$) for four hours, or 0.25 ppm (655 $\mu g/m^3$) for eight hours. These studies extended over a ten-year period and involved over 300,000 hours of air sampling. During this sampling period, a correlation was made among ground-level average SO_2 concentrations occurring during daylight hours, duration of the fumigation, and the species of plants growing near the recorders that responded to the fumigation. All significant fumigations that occurred were given an intensity rating, and the plant species that developed acute markings from each fumigation were noted. The intensity ratings were arrived at by multiplying the average ppm SO_2 for one hour by 105, or two hours by 182, or four hours by 286, or eight hours by 400.

From such work as O'Gara's and the Canadian studies, sensitivity tables have been constructed (Table 4-4). These are quite useful when observing suspected SO_2 injury in the field.

Table 4-4

Relative Sensitivity of Plants to SO_2

Crops

Sensitive		*Tolerant*
Alfalfa	Sweet Potatoes	Celery
Buckwheat	Lettuce	Corn
Barley	Beans	Onion
Cotton	Broccoli	Potato
Red Clover	Squash	Cabbage
Oats	Wheat	
Peas	Carrot	
Spinach	Tomato	

Flowers

Sensitive		*Tolerant*
Sweet Pea	Sweet William	
Four O'Clock	Gladiola	
Cosmos	Tulip	
Bachelor Button	Violet	
Aster	Zinnia	

Trees

Sensitive		*Tolerant*
Trembling Aspen	Alder	Cedar
Jack Pine	Red Pine	Citrus
White Pine	Austrian Pine	Maple
White Birch	Hazel	Linden
Larch	Apple	
Large-Toothed Aspen	Douglas Fir	
Willow	Ponderosa Pine	

Garden Plants

Sensitive		*Tolerant*
Peas	Cucumber	Muskmelon
Rhubarb	Lettuce	Corn
Spinach	Radish	Onion
Swiss Chard	Squash	Potato
Beans	Tomato	Cabbage
Beets	Cultivated Mustard	Broccoli
Turnips	Kohlrabi	Turnips
Carrot		

Table 4-4 (Continued)

Relative Sensitivity of Plants to SO_2

Miscellaneous		
Sensitive		*Tolerant*
Prickly Lettuce	Sow Thistle	Privet
Ragweed	Yellow Clover	Lilac
Dock	Night Shade	
Bracken Fern	Chickweed	
Wild Mustard	Annual Bluegrass	
Careless Weed	Brazilian Pepper Bush	

Air Quality Standards

To protect man and his possessions against injury from SO_2 fumigations, primary and secondary standards have been set by the Federal Government. The primary standards, effective April 30, 1971, require that the annual SO_2 average in the atmosphere not exceed 0.03 ppm (80 $\mu g/m^3$). In addition, an average concentration of 0.14 ppm (365 $\mu g/m^3$) of SO_2 for a 24-hour period is not to be exceeded more than once a year. A secondary standard has also been set, requiring that a three-hour average of 0.5 ppm (1,300 $\mu g/m^3$) not be exceeded more than once a year.

Relative Sensitivity of Plants to SO_2

Plants vary widely in their sensitivity to SO_2. Alfalfa and barley are usually considered to be among the most sensitive. However, some of the common weeds, such as the velvet-weed, dock, mallow, and prickly lettuce are nearly as sensitive. In addition, these plants are so sensitive that they must appear in the same grouping: zucchini and yellow summer squash (Plate 4-18), trembling aspen (Plate 4-4), white birch (Plate 4-3), bracken fern (Plate 4-1), tulip (Plate 4-19), wild geranium, buckwheat, red clover, jack pine, white pine, sow thistle, and Brazilian pepper bush.

It would appear that in the Sudbury area some injury to the most sensitive plant species could occur without the secondary standard being exceeded. In this area, large quantities of SO_2 are emitted daily into the atmosphere from smelter stacks that are not considered to be really tall, compared with recent installations. Thus, average and short duration ground levels of SO_2 might be high in this area. It should be remembered that plants on high-sulfur nutrition are more sensitive to SO_2 fumigations than are comparable plants on a normal sulfur diet. Therefore, plants in a high SO_2 environment might be more responsive to SO_2 than plants in less contaminated areas.

Leaf Tissue Analysis

Sulfur is an essential plant nutrient and as such accumulates in plant tissues whether taken in from the soil or air. Therefore, analyzing the amount of sulfur in leaves provides a rough approximation of the amount of sulfur to which those leaves have been exposed. Because of this accumulation, some investigators have tried to use the sulfur content of leaves as a measure of sulfur-dioxide contamination. Unfortunately, as a normal and important constituent of plant proteins, sulfur is already present in fairly high concentrations (roughly about 0.1 to 0.2 percent of the dry weight), so that further increases originating from SO_2 tend to be obscured. The only real value from sulfur analysis comes when background levels in the foliage are already known. Even then, analysis provides only a general idea of the degree of exposure. It does not reveal the duration of exposure or the concentrations of SO_2 to which the plant was exposed. Most importantly, it does not disclose the extent of toxicity or injury from the SO_2.

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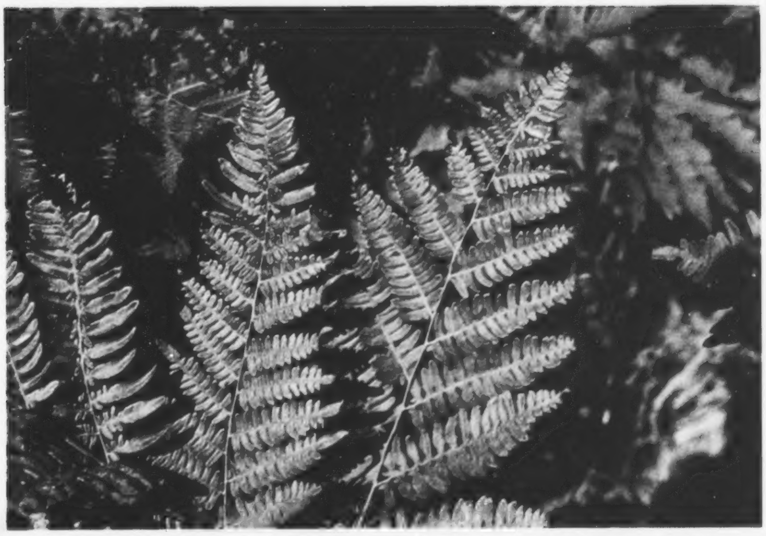

Plate 4-1. *Acute Sulfur Dioxide Injury to Bracken Fern. Typically Orange-Red; Occurs on Margins of Fronds*

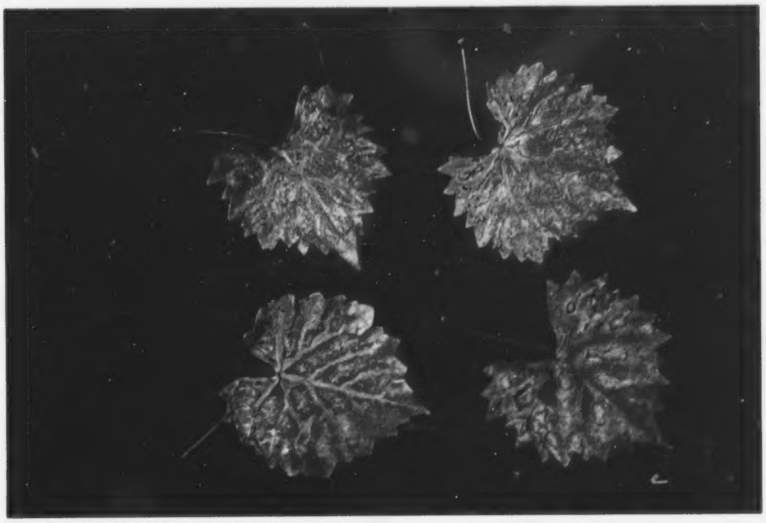

Plate 4-2. *Typical Sulfur Dioxide Injury to Fox Grapes. Light Brown, Occurs Between Veins; Top Leaves Show Undersurface, Bottom Leaves Show Upper Surface*

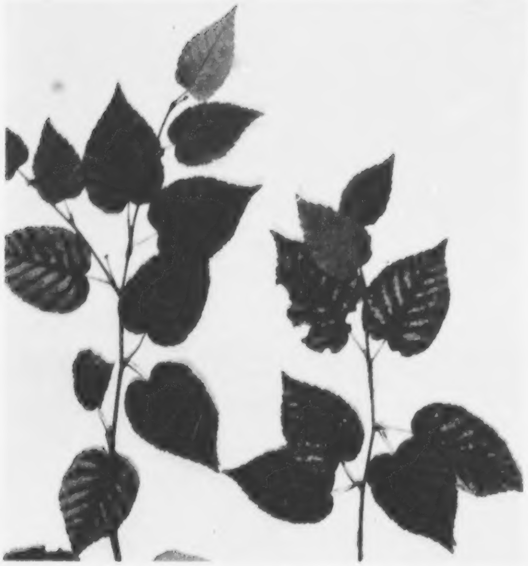

Plate 4-3. *Acute Sulfur Dioxide Injury to White Birch Foliage*

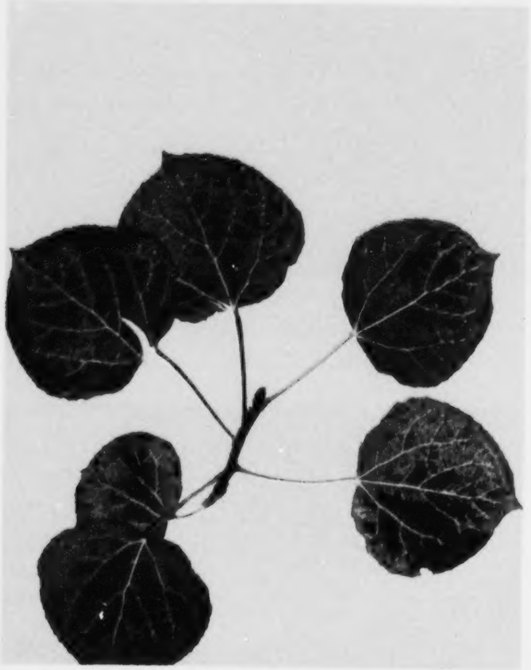

Plate 4-4. *Acute Sulfur Dioxide Injury to Trembling Aspen Leaves. Injured Areas are Reddish-Brown and Darken with Age*

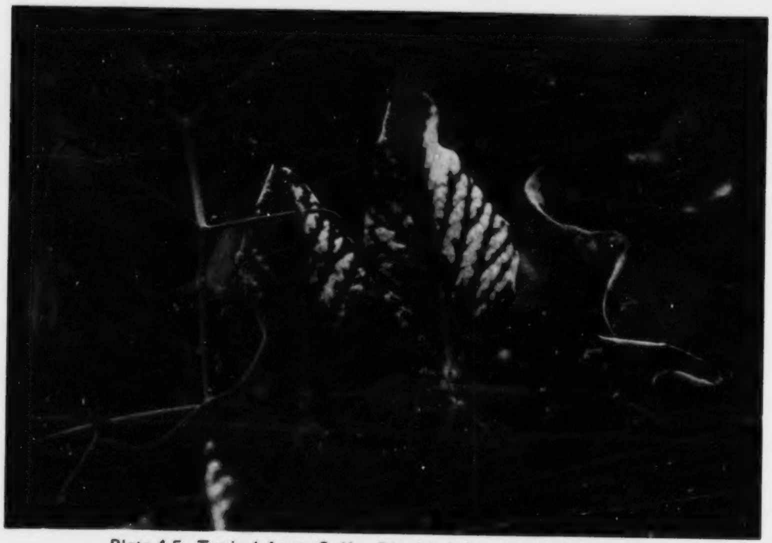

Plate 4-5. *Typical Acute Sulfur Dioxide Injury to the Garden Pea*

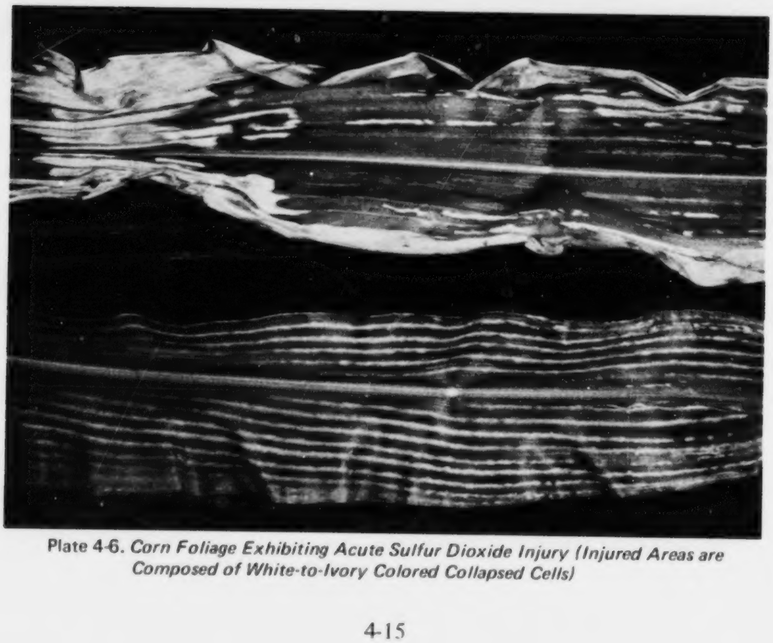

Plate 4-6. *Corn Foliage Exhibiting Acute Sulfur Dioxide Injury (Injured Areas are Composed of White-to-Ivory Colored Collapsed Cells)*

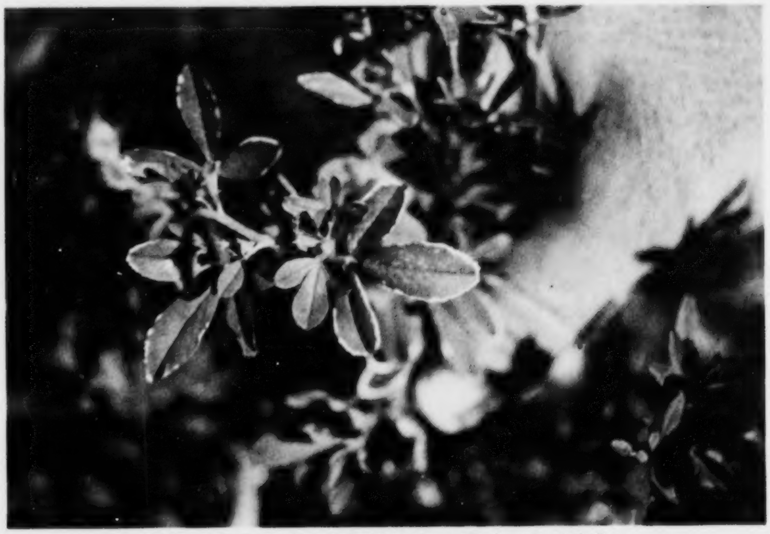

Plate 4-7. *Light Acute Sulfur Dioxide Injury to Alfalfa*

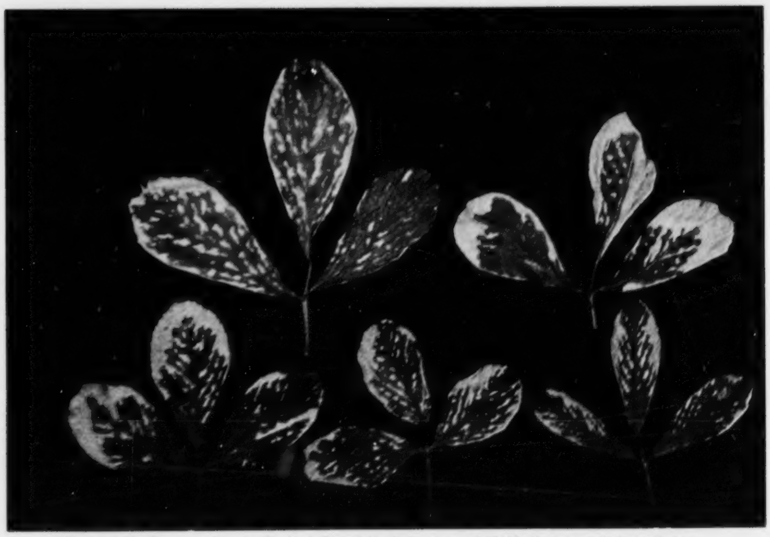

Plate 4-8. *Severe Acute Sulfur Dioxide Injury to Alfalfa Leaves. Injury Ivory Colored, Marginal with Intercostal Spotting*

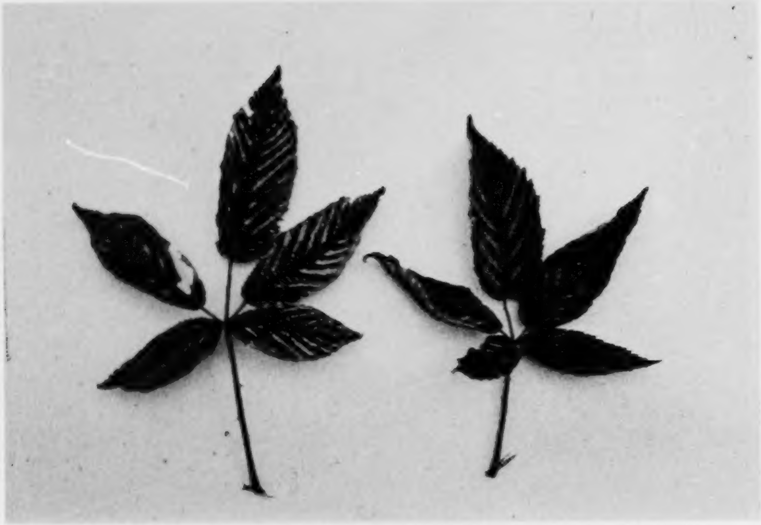

Plate 4-9. *Acute Sulfur Dioxide Injury on Blackberry Growing Within Five Miles of the Source*

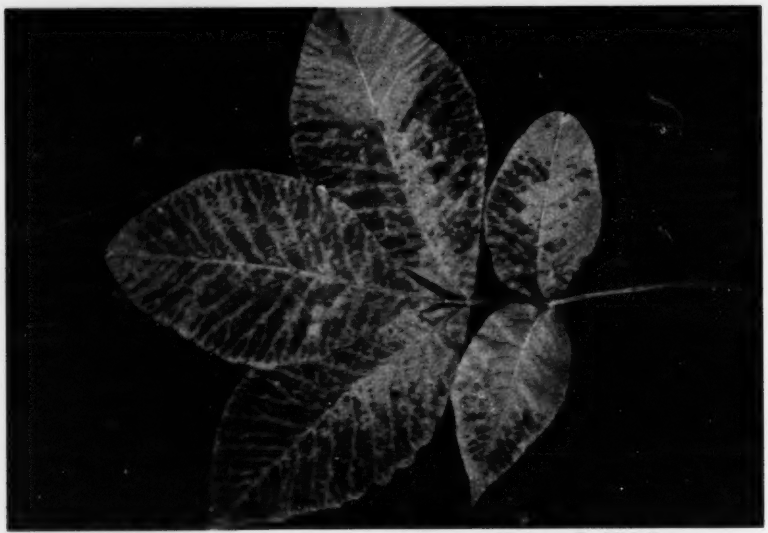

Plate 4-10. *Typical Acute Sulfur Dioxide Injury to Hickory. Reddish-Brown; Frequently Extends from Near Midrib to Near Edge of Leaf*

Plate 4-11. *Acute Sulfur Dioxide Injury to Eggplant Leaves (Ivory-to-Light Tan)*

Plate 4-12. *Acute Sulfur Dioxide Injury to Quince Foliage (Colorful Brownish-Red)*

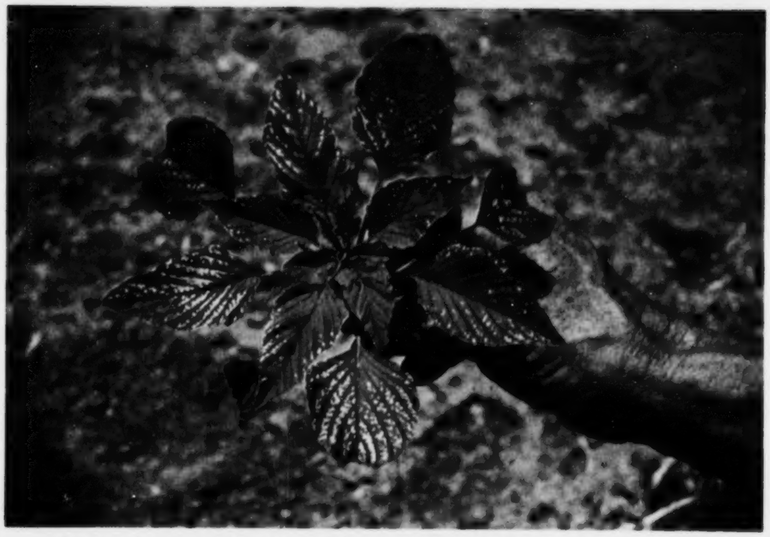

Plate 4-13. *Acute Sulfur Dioxide Injury to Careless Weed (Ivory-Colored; Note Little or No Injury on Young Expanding Leaves)*

Plate 4-14. *Chronic Sulfur Dioxide Damage to Cotton Foliage in Palm of Hand. Note Injured Areas are Chlorotic But Not Collapsed; Veins Usually Remain Green*

Plate 4-15. *Acute Sulfur Dioxide Injury to Sonora Wheat (Ivory-Colored)*

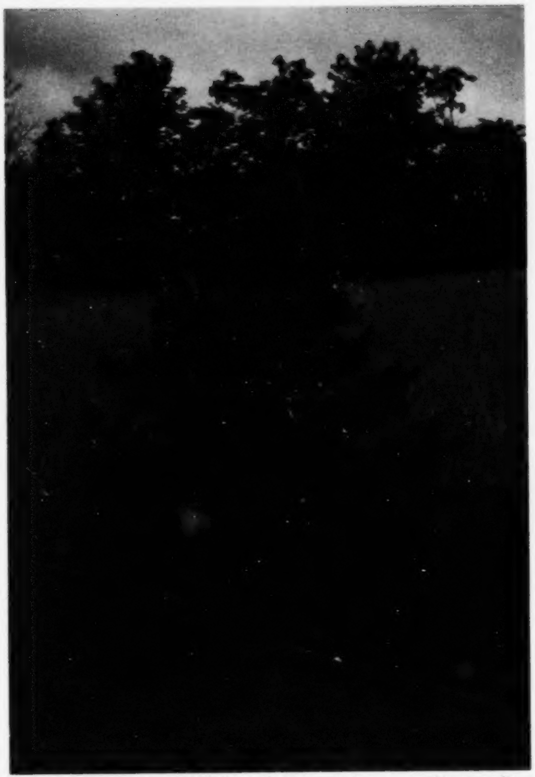

Plate 4-16. *Suspected Sulfur Dioxide Injury on Virginia Pine Growing 1.5 Miles from a Source*

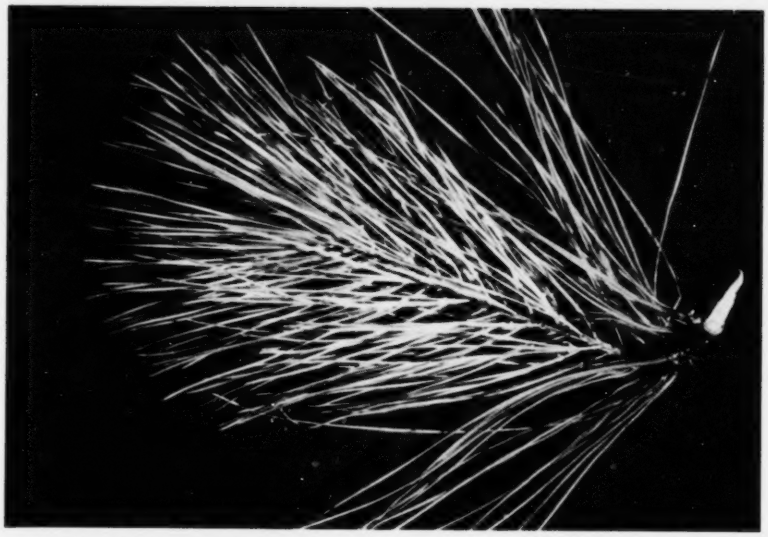

Plate 4-17. *Typically Acute Sulfur Dioxide Injury to Loblolly Pine Needles*

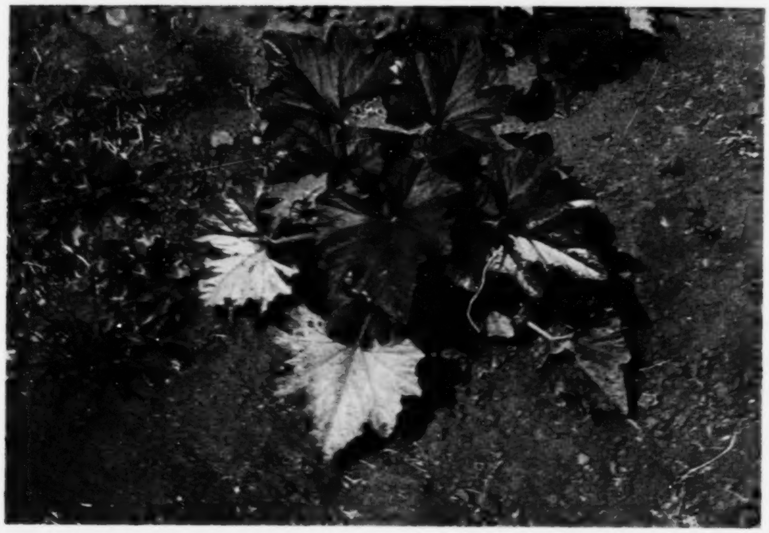

Plate 4-18. *Acute Sulfur Dioxide Injury to Yellow Summer Squash. Acute Injury to all Squash and Pumpkin Plants is White or Ivory-Colored*

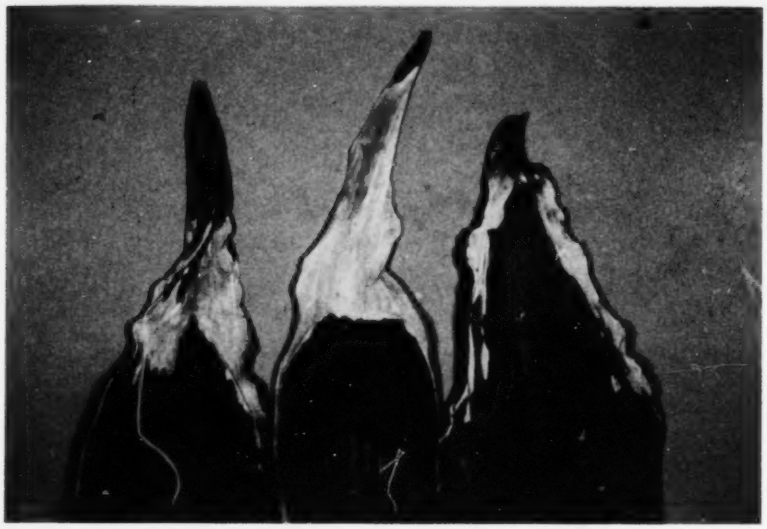

Plate 4-19. *Acute Sulfur Dioxide Injury to Tulip. Note Ivory-Colored Injury Occurs at Leaf Tips and Margins*

Chapter 5

FLUORIDES

Introduction

Fluorine is a relatively common and abundant element of the earth's crust. It is present in soils as fluoride, and in minerals, such as fluorspar, biotite, apatite, hornblende, and muscovite. In one ton of soil, on the average, there is about four-tenths of a pound of fluoride or 190 parts per million (ppm). It has been estimated that about 30 million tons of soil enter the atmosphere of the United States each year and this is equivalent to about 6,000 tons of fluoride per year as the rate of emission for one natural source. By comparison, it was estimated that industrial emissions in the United States totaled 119,000 tons of fluoride for 1968.

Atmospheric fluoride had adverse effects on agriculture as a natural pollutant long before it became a man-made one. Air-borne fluoride sometimes affected agriculture adversely without directly affecting plants. Livestock in parts of North Africa developed an illness called "darmous" when prevailing winds carried a relatively fluoride-rich fluoroapatite dust onto vegetation. This illness was due to fluorosis that occurred when the vegetation was consumed by the animals. Volcanoes and fumaroles are another natural source of gaseous and particulate atmospheric fluoride. Roholm has written, "In the Icelandic literature from round about the year 1000 up to recent times we find accounts of how the domestic animals turned sick and died when there were volcanic eruptions . . . the disease attacked animals which ate the grass contaminated with the fallen ash, which means sheep especially, as cattle and horses were kept stabled as much as possible and so escaped [illness] . . ."

It is not known exactly when man's activities first generated atmospheric fluoride to the extent that it adversely and directly affected plants. Reports of fluoride-induced injury to vegetation began to appear in the late 19th century with the advent of certain industrial processes and became more frequent as new processes developed and others expanded. In general, man's activities can be viewed as gradually augmenting and changing the natural rates at which fluoride is transferred among earth, air, water, and organisms; a composite picture of environmental fluoride is given in Figure 5-1.

Sources

Atmospheric fluoride reaches the plant in the form of a gas, particulate matter, or gasecus fluoride adsorbed to particles. The most phytotoxic and best studied form of atmospheric fluoride is gaseous hydrogen fluoride (HF). Al-

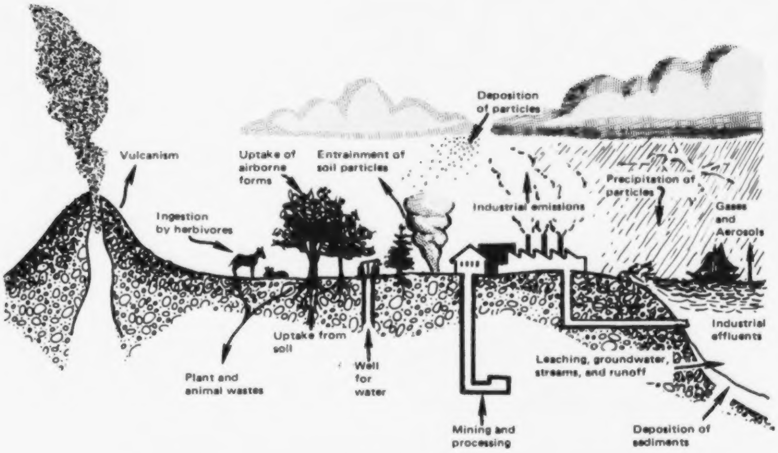
Deposition of particles
Vulcanism
Uptake of airborne forms
Entrainment of soil particles
Industrial emissions
Precipitation of particles
Ingestion by herbivores
Gases and Aerosols
Industrial effluents
Uptake from soil
Leaching, groundwater, streams, and runoff
Plant and animal wastes
Well for water
Mining and processing
Deposition of sediments

Figure 5-1. *Composite Picture of Environmental Fluoride*

Source: *Fluorides.* Washington, D.C.: Committee on Biological Effects of Atmospheric Pollutants, National Academy of Sciences, Publication ISBN O-309-01922-2, 1971.

though silicon tetrafluoride (SiF_4) may be evolved, atmospheric reactions probably convert it to HF before it reaches the plant. The particulate forms of fluoride have not been so extensively studied as the gaseous forms with respect to their effects on plants. In general, they exist as salts and their toxicity may be determined by the characteristics of the particles and the environmental conditions that affect their collection and retention by foliage and the subsequent penetration of fluoride into the foliar tissue. However, when forage crops or vegetation that is eaten by wildlife is of concern, the presence of particulate material on the foliage must also be considered.

Any process that utilizes fluorine-containing material can be a potential source of atmospheric fluoride. Fluorides may be emitted when fluorine-containing material is ground, crushed, or pulverized; treated with strong acids; heated; or used in some process in a gaseous or volatile form. The magnitude of the source depends upon the kind of process, kind of material, volume of material processed, and degree of emission control. An estimate of the quantities of fluorides emitted by different industries is given in Table 5-1.

The emission of fluorides can result from the manufacture of phosphate fertilizers and feeds, phosphoric acid, or elemental phosphorus. The phosphate rock used as a raw material in these industries contains 3 to 4% fluorine. Dust can be produced in the grinding and drying of the rock, and gaseous fluoride can be produced (as HF or SiF_4) when the rock is treated with sulfuric acid to produce phosphates or is heated in electric furnaces for the production of elemental phosphorus. The ponds that contain waste process water may also be significant sources of atmospheric fluoride.

Table 5-1

Estimated Total Fluoride Emissions from Major Industrial Sources in the United States in 1968

Source	*Atmospheric Emissions, tons/year*
Manufacture of brick and tile products	18,500
Manufacture of steel (open-hearth furnace)	16,800
Manufacture of aluminum	16,000
Combustion of coal	16,000
Manufacture of steel (electric furnace)	14,900
Manufacture of normal superphosphate fertilizer	9,700
Manufacture of steel (basic-oxygen furnace)	8,400
Manufacture of elemental phosphorus	5,500
Nonferrous-metal foundries	4,000
Manufacture of wet-process phosphoric acid	3,000
Manufacture of glass and frit	2,700
Welding operations	2,700
Manufacture of triple superphosphate fertilizer	300
Manufacture of diammonium phosphate fertilizer	100
Manufacture of phosphate animal feed	100
TOTAL	118,700

Source:
Fluorides. Washington, D.C.: Committee on Biological Effects of Atmospheric Pollutants, National Academy of Sciences, Publication ISBN 0-309-01922-2, 1971.

In the manufacture of aluminum, alumina is dissolved in molten cryolite and reduced electrolytically. Both cryolite and fluorite, which is also present in the process, contain fluoride, and in the electrolyte cell fluorine is evolved in gases such as hydrogen fluoride, or as particulate matter such as fluoride salts. In steel-making, fluorspar is used as a flux from which hydrogen fluoride may be generated. The combustion of coal can be another source of atmospheric fluoride because coal contains an average of 0.008% fluoride. The clays used for the manufacture of bricks, pottery, and cement contains fluorine, and gaseous fluorides may be emitted from the kilns. In the manufacture of glass and enamel, fluoride-containing materials that are heated evolve gaseous and particulate fluorides. Other sources of fluorides are industries that produce hydrogen fluoride and those that use it for petroleum refining or the production of fluoro-carbons.

Atmospheric Chemistry

There is no general set of concentrations that can be expected for all fluoride sources. The extent and magnitude of atmospheric concentrations over the area affected will depend upon the rate of emissions. Most major sources are characterized by a relatively constant rate of emission. Therefore, pattern of dispersion, in time and space, will be determined by the combination of source, topography, and meteorology. Diurnal and seasonal fluctuations can be expected to follow the changes in those climatic and meteorological factors that determine dispersion. Occasional high concentrations (relative to the mean or median) may occur due to failure of controls, accidental spills, or unusual meteorological conditions.

Monitoring Data

In contrast to the better studied urban and industrial air pollutants – photochemical oxidants and sulfur dioxide – there is no extensive compilation of ambient levels of atmospheric fluoride. The lower limit of detection may range from 0.01 to 0.05 $\mu g/m^3$ (about .012 to .06 ppb)* in a 24-hour sample depending upon the methods used. The lower limits for shorter sampling periods are higher.

The analysis of more than 7,700 air samples for total water-soluble fluoride by the National Air Pollution Control Administration over the years 1966 and 1967 showed that 97% of the samples from non-urban areas had no detectable fluoride. The highest concentration was 0.16 $\mu g/m^3$ (.195 ppb). In urban areas the corresponding values were 87% and 1.89 $\mu g/m^3$ (2.306 ppb). In the heavily industrialized Ruhr area of Germany, samples in the city of Duisburg during 1965 and 1966 showed a mean of 1.3 $\mu g/m^3$ (1.586 ppb).

Symptomatology

The HF-induced symptoms that appear on the foliage of plants result from a series of complex events: the interaction of fluoride and metabolic processes in the leaf and the action of environmental factors upon it. Some general aspects of symptom development will be considered here so that the symptoms and their variations can be more easily described and understood. Specific guidelines for diagnosing fluoride-induced symptoms are presented in Chapter 10.

Of what do symptoms consist? First, there is a change in the color of a portion of the leaf due to a loss of chlorophyll in the affected area. When the amount of this pigment is reduced, the yellow pigments that remain are unmasked and a condition of chlorosis, which may range from a slight paling of the green to an appearance of a yellow color in the affected area, appears. Second, a color change may be due to death of the tissue and the formation of brown pigments as the affected area dies. To a certain extent, the coloration of this necrotic area depends upon the species and how rapidly the necrosis develops. Third, symptoms may consist of deformation as well as discoloration of the foliage. These deformations occur usually on broad-leaved plants as a result of necrosis which occurs as the leaf is expanding. The tip of the leaf may become necrotic and the rest of the leaf may become cupped as it expands, or the tissue between the veins may expand more rapidly than the veins, giving the leaf a savoyed appearance. Sometimes the edges of the leaf take on a ragged appearance owing to the loss of necrotic marginal tissue.

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*ppb = parts per billion, in which "billion" means "thousand-million" or 10^9 and not 10^{12} as in British and German usage. American and French usage agree however. See Appendix A for methods of conversion between ppm and $\mu g/m^3$. One ppb is 10^{-3} ppm. Atmospheric concentrations for HF use ppb to avoid awkward numbers. The unit ppb refers only to atmospheric concentration and is v/v.

Where do symptoms occur? The distribution of the affected areas, or lesions, on the leaf is usually determined by two factors: (1) the species of plant, which determines the morphology of the leaf; (2) the severity of the exposure, which determines the response of the leaf. Of these two factors, species as related to leaf morphology is probably the more important. Generally, the lesions induced by fluoride appear first or are more intense in the apical and marginal areas of the leaf. Young expanding leaves are most likely to be injured by an exposure. Exceptions to these generalizations occur and will be discussed later.

How does fluoride produce these symptoms? The mode of action of fluoride is imperfectly understood, but fluoride is known to act as an inhibitor of plant metabolism. As fluoride accumulates in the leaf, it causes an increased inhibition of metabolism, and chlorosis appears as the first visible sign of this metabolic disruption. With further increases in the concentration of fluoride in the tissue, the cells die and necrosis is observed. When the leaf is exposed to relatively high concentrations of HF for short periods of time, local concentrations of fluoride in the leaf may rapidly exceed the toxic threshold, and irregular patches of necrosis may occur in the intercostal areas. But more often, the leaf is exposed over a longer period of time, constantly or periodically, to relatively low concentrations. Under these conditions, the fluoride absorbed by the leaf is translocated to and accumulates in the apical or marginal tissues where the more characteristic symptoms of fluoride toxicity appear.

The short-term, high-level and the long-term, low-level exposures are commonly referred to as acute and chronic, respectively. Whereas acute exposures usually produce necrotic lesions, chronic exposures may produce either necrosis or chlorosis depending upon the species and tolerance of the plant. Therefore, it matters not so much whether a symptom be classified as acute or chronic but what the severity of the symptom, its distribution on the plant, and its occurrence on different species tell about the kind of exposure that has occurred.

Broad-leaved Plants

Within this group of plants, symptoms consist of chlorosis of varying intensity, or necrosis, or both. The affected areas are initially located at the tips or margins of leaves and, with increasing severity, extend toward the base of the leaf from the tip or toward the middle of the leaf from the margins.

Citrus. In naval orange, irregular chlorotic patches appear in the intercostal areas of the leaf near the margin and tip. With prolonged exposure, these patches extend further towards the midrib and base and increase in intensity until the midrib and venation appear as a green-arborescent figure on a pale chlorotic field (sometimes called the "Christmas tree" pattern of chlorosis). Necrosis occurs principally at the tips of nearly mature leaves with a chlorosis and mottling which affect the remaining portion but decrease in intensity toward the base of the leaf. In lemon, chlorosis develops also along the margins and tips of leaves and with increasing severity involves the intercostal areas to give the "Christmas tree" pattern of chlorosis. In grapefruit, symptoms may first appear as irregular, ill-defined chlorotic blotches in the intercostal areas or as a thin marginal area of chlorosis. With increasing severity the marginal and interveinal chlorosis intensifies and the affected areas merge (Plate 5-1). This pattern is most common on older leaves.

Some of the chlorotic and necrotic symptoms of citrus foliage resemble injury from excessive boron, but a gumming of the undersurface of the leaf is found when boron toxicity is responsible. The "Christmas tree" pattern of chlorosis also resembles that induced by manganese deficiency.

Other fruit crops. On grape (European) necrosis may appear along the margins of the leaf and extend irregularly into the intercostal areas, with some zonation or banding of the necrotic areas.

On blueberry (Plate 5-2) necrosis of the apical and marginal portions of the leaf can develop under chronic exposure. With low bush blueberry, a reddening of the tip and apical margins of the leaf can precede the development of necrosis in chronic exposures. However, this reddening can also be induced by other conditions.

Apple leaves show a very dark-reddish brown coloration at the tip and margin of the leaf with very little necrotic flecking of the interveinal areas. Grape leaves (Plate 5-3) show necrosis of both the marginal and intercostal tissues after more acute exposure. Injured areas are initially dull or pale green, then change to buff or light tan with death and desiccation. Frequently, these dry, brittle portions fall away from the uninjured portion of the leaf, leaving an irregular margin. Prune foliage shows about the same pattern of injury as apricot, but the necrotic areas may be colored a much darker brown. If recurrent injuries occur, the necrotic areas show zonation or bands of darker necrotic tissue within the paler necrotic areas. On peach and sour cherry foliage, necrosis in trace amounts will appear at the tip of the leaf. The damaged area may fall from the leaf leaving a notched leaf tip. If this kind of injury occurs before the leaf has completed expansion, a cupping of the leaf may occur. On the foliage of apple, sour cherry, and peach, chlorosis may appear on the tips and margins of the leaf after chronic exposures. Intensification of this symptom results in more pronounced chlorosis and an expansion of the chlorosis into the intercostal areas.

As with citrus, certain nutrient deficiencies, such as zinc or manganese, can also induce the "Christmas tree" pattern typical of chlorosis. Low temperature and drought injury, oil sprays, and virus diseases can induce fluoride-like symptoms.

Other Deciduous Trees and Shrubs. The range of symptoms seen on deciduous trees follows the same patterns seen on other broad-leaved plants (Plate 5-4). Acute exposures may cause necrosis of the tips and margins and sometimes of intercostal areas of leaves with irregular but distinct demarcation between dead and live tissue. Chronic exposure will tend to cause chlorosis, first of the tips or serrations, then of the apical and marginal areas, and finally of the intercostal areas. The venation of damaged intercostal tissue is a darker green than the laminar tissue. Sometimes chronic injury may appear only as a necrosis of the tips of lanceolate leaves or tips of lobes of palmate leaves, such as maple. On some species, such as poplar, intercostal chlorosis may accompany marginal necrosis.

To the same extent as with other groups of plants, the kind of exposure, kind of leaf, and tolerance of the plant will determine the kinds of symptoms. Moreover, natural or other kinds of stresses can induce symptoms that mimic those induced by atmospheric fluoride (Plate 5-5).

Herbaceous Weeds. St. John's wort is one of the more ubiquitous and sensitive species of herbaceous weed. Fluoride-induced injury appears on it as a red-brown necrosis of the apical portion of the leaf. Chronic exposure produces a light-green flecking and then chlorosis on the younger, expanding leaves of lamb's-quarters (Plate 5-6). More intense symptoms appear as an increase in the intensity of chlorosis, chlorosis of the intercostal areas, and a cupping and puckering of the leaves.

In chronic exposures, chlorosis develops at the tips and margins of the expanding leaves of night-shade, knotweed, mares tail, and crab grass. When necrosis of leaf tips occurs, relatively little chlorosis precedes it. Pigweed, smartweed, ragweed, golden rod, and oxalis also show these kinds of symptoms. In acute exposures, apical markings are more common on chickweed, nettle-leaf goosefoot, and blue grass; marginal markings are more common on mustard, sunflower, dandelion, and cheeseweed; and both kinds of markings appear on pigweed.

Narrow-leaved Plants

There is probably no single type of symptom that would apply to all monocots, but there are certain kinds of symptoms that are typical for many species.

Grains and Cane. Sweet corn is a reasonably ubiquitous and sensitive indicator of atmospheric fluoride, although sensitivity depends upon the cultivar (Plate 5-7). Fluoride-induced symptoms usually can be distinguished from those caused by disease, insect injury, mineral deficiencies, or other pollutants by appearance, pattern, and development. At the lowest exposures that produce symptoms, there are scattered chlorotic flecks or stipples near the tip of the leaf and along the apical margins of the leaf. With an increase in exposure, the area affected by stippling extends down toward the base of the leaf along the margins and inward on the leaf toward the midrib. The intensity of the stipple diminishes toward the basal or medial areas of the leaf. Also, with an increase in severity, the stippling along the apical margins will increase and coalesce to give a chlorotic band of tissue at the tip and along the margins. As with the stipple, this band of chlorosis decreases in width toward the base of the leaf. With a further increase in severity, necrosis appears at the tip and then spreads along the margins of the leaf toward the base and inward toward the midrib.

The symptoms produced by fluoride in milo maize (sorghum) are similar to those of corn. The effect starts at the tip and the immediately adjacent margins of the leaf. As the effect increases, the affected area enlarges by an extension of the marginal areas down the leaf and toward the midrib. Lesions in the affected area start as chlorotic stipples, coalesce into chlorotic bands, and may finally become necrotic with increasing severity of exposure. In milo maize, irregular red areas may border the first observed necrotic areas. The patterns and severity of symptoms tend to be more intense on the older leaves if exposure occurs around the time of heading.

In grasses and small grains, fluoride symptoms may sometimes appear as chlorotic stipples or chlorosis on the tips and margins of leaves. But most often they appear as a necrosis of tissue at the apical portion of the leaf. In barley, the apical portions of the awns may become necrotic if exposure occurs during the later phases of head development or maturation.

Injury to sugar cane by HF shows about the same pattern and sequence of chlorosis and necrosis as injury to sorghum and corn leaves.

Several climatic and environmental conditions can produce symptoms in grains and other grasses that mimic fluoride-induced injury. Hot winds can cause terminal bleach of cereals. Subfreezing temperatures may cause a marginal necrosis and red discoloration on milo maize that is similar to HF-induced necrosis, except that a silvering of the upper surface at the arch of the leaf may frequently be present. Some mineral nutrient deficiencies in corn, such as zinc or potassium, can produce a pattern of chlorotic and necrotic banding on the leaves; however, the distribution of the symptom over the plant is different from that induced by HF. Similarly, mite injury or genetically-induced mottling may have a different appearance or distribution over the leaves of the corn plant than fluoride-induced injury.

Gladiolus and Similar Plants. The most popular biological indicator of fluoride effects is gladiolus; more data exist, and more observations are made in the field, for this species than for any other. The typical symptom of fluoride-induced injury on gladiolus is necrosis of some apical portion of the leaf. With most kinds of exposure in the field, necrosis starts at the apex of the leaf and proceeds downwards toward the base (Plate 5-8). The color of the necrotic area depends upon the variety of gladiolus and ranges from ivory or light tan to various shades of brown. Generally, light-flowered varieties show the lighter shades. The necrotic area is sharply delineated from live tissue, usually by a dark brown band of necrotic tissue at the base of the necrotic area and sometimes by a very narrow band of chlorotic tissue below the necrosis. An evenness of the band or front of necrosis across the leaf indicates a relatively constant chronic exposure. The length of tipburn is often used as a quasi-quantitative measure of the dosage received by the plant. Variations in this type of symptom can occur. With acute dosages, the line separating necrotic and live tissue, although sharp, tends to become uneven and may extend further along one margin than the other. With high concentrations of HF, isolated necrotic areas may appear along the margins or between the veins (Plate 5-9). When several exposures have occurred, dark brown bands may be seen in the necrotic portion of the leaf. Each band marks the increments of necrosis produced by successive exposures.

The flowers of gladiolus are more tolerant to fluoride than the foliage, but bracts are relatively sensitive and injury appears as a necrosis that extends toward the base of the bract from the tip. When injury occurs on flowers it appears first as a watersoaked tissue at the margins of the sepals and petals. This tissue then dries and becomes necrotic.

Tulips (Plate 5-8), iris, lilies, and similar monocots show HF-induced symptoms that appear as a necrosis that starts at the leaf tip and extends along the margins and toward the base of the leaf.

Several agents can produce fluoride-like symptoms on gladiolus and similar plants. Water stress produces tip and marginal necrosis of the flower bracts that can be indistinguishable from fluoride-induced injury. When foliar necrosis is induced by moisture stress, zonations in the necrotic area may be lacking, and there may not be a sharp demarcation between dead and live tissue. Botrytis lesions on gladiolus may be distinguished by their dull color and indefinite margins; severe leaf tip necrosis is accompanied by many lesions, 1/16 to 1/4 of an inch in diameter, which are not confined to the marginal and apical portions of the leaf. Finally, gladiolus may show a fluoride-induced necrosis that results from fluoride in the soil, not the air. The occurrence and severity of this injury depends upon the fluoride content of the fertilizer, its rate of application, and the kind of soil.

Coniferous Plants

The typical fluoride-induced symptom on the foliage of conifers is a necrosis of the apical portion of the needle. It is often called "needle scorch" or "tipburn" (Plate 5-10). The boundary between dead and live tissue is even and well defined, and the band of necrotic tissue next to the live portion may be a darker shade of brown than the adjacent necrotic tissue. The percentage of the total length of the needle that is necrotic usually increases with the severity of the exposure and may involve only a few millimeters at the tip, to the tip-half, or almost the entire needle. On many conifers, the necrotic portion is a reddish-brown color, and, if on the current year's needles, fairly constant in color (Plate 5-11). If the needle is observed immediately after an injurious exposure has taken place, the apical tissue may have a pale gray-green appearance. After another time period, this tissue will have become necrotic and the boundary between necrotic and healthy tissue may have this same gray-green appearance as additional necrotic involvement occurs. The appearance of a slightly darker brown band delimiting the necrotic and healthy tissues may be the final stage of symptom development. Another portion of the needle, immediately below the necrotic area, may turn necrotic if a subsequent exposure occurs. Thus, when several episodes have occurred, one may see more than one band in the necrotic tissue, each corresponding to the appearance of another increment of necrosis.

In general, there are three kinds of exposures that can result in an easily discernible amount of necrosis on the needle: a single acute exposure; several not so acute exposures; and continuous or intermittent exposures over an extended period of time with necrosis appearing with the onset of some period of environmental stress. Although one can distinguish between several occurrences of necrosis and a single one upon examination, the distinction between necrosis due to a single acute exposure and that due to prolonged, chronic exposure is made mainly by repetitive examination of the vegetation through a growing season.

Chlorosis, if it appears, may only involve the one to two millimeters of the needle's tip. Color may vary with the species. For example, necrotic areas of blue spruce appear to be purple.

Fruit

Suture red spot (or "soft suture") is a fluoride-induced syndrome of peach fruit, which was first noted when fluoride-containing sprays were tested for the control of insects. It has since been produced by experimental fumigations of peach trees with HF. This syndrome is characterized by the premature ripening of the flesh along both sides of the suture towards the stylar (terminal) end of the fruit (Plate 5-12). Externally, the skin over the affected tissue shows a reddening; internally (in yellow-fleshed varieties), a wedge of red tissue extends from the skin towards the pit in clear contrast to the paler unaffected tissue. By the time the rest of the fruit ripens, the affected tissue becomes soft or rotten. Sometimes the flesh may separate along the suture or the pit may split. Although enlargement or swelling along the suture may accompany discoloration of tissue, hypertrophy of the affected tissues is not common. The concentrations of gaseous fluoride that can induce suture red spot on fruit are less than those that induce foliar symptoms.

A premature ripening of the suture of peach fruit can have other causes. Growth regulants, such as the phenoxyacetic acids (e.g., 2,4-D), which may come from neighboring agricultural operations, induce a premature ripening of the suture, but the effect is most severe towards the stem end of the fruit and hypertrophy of the affected tissues occurs. This hypertrophy may affect one side of the suture more than the other. Premature ripening of the suture has also been attributed to virus infection, aberrant fruit development, and unknown agents.

Symptoms similar to suture red spot have been described on apricot, cherry, and pear fruits and attributed to atmospheric fluoride. They may consist of a necrosis of tissue or premature ripening and shrivelling of tissue at the stylar end of the fruit. Similar symptoms on these fruits also have been associated with moisture stress.

Factors Affecting Plant Response

General Characteristics

The factors that affect the plant's response to atmospheric fluoride can be considered in several different ways. First, these factors can be categorized by their nature and origin as follows:

1. Biological characteristics of the plant, including its genetic complement, and stage of development;
2. Environmental conditions, such as climate, light quality and intensity, temperature, relative humidity, precipitation, soil conditions and

presence of pathogens or insects;

3. Pollutant characteristics, including physical and chemical forms of fluoride, concentration and duration of exposure, and frequency of exposure periods.

Second, their mode of action on the plant can be postulated as an effect on:

1. Absorption or penetration of fluoride into the foliar tissue;
2. Accumulation of fluoride in an active form at a sensitive site or tissue;
3. Metabolic and physiological state of a tissue that determines its sensitivity to the toxic action of fluoride.

Third, the operation of these factors can be separated into three phases:

1. Before exposure occurs
2. At the time of exposure
3. After exposure has occurred

Because the primary objective is to diagnose and assess the plant response to fluoride in the field, certain aspects of these factors and their actions are emphasized while others receive lesser attention.

Genetic and Specific Differences in Tolerance

Variations in plants' genetic tolerance to fluoride-induced injury are greatest among different species; however, differences in tolerance among varieties are better indicators of the role that the genetic complement of a plant plays in determining its tolerance to HF. Various cultivars of corn (Plate 5-7), sorghum, gladiolus, and tomato, and the variability of individuals in populations of conifers (Plate 5-13) display marked differences in their tolerance to HF. It is not known how many genetic factors are involved in tolerance.

Stage of Development

As has been mentioned before, the young elongating or expanding foliage of plants is the tissue most sensitive to HF. In an indeterminate plant, in which shoot growth and leaf initiation occur throughout the growing season, sensitive tissues are present throughout the season. In plants, such as pine or oak, the sensitivity of the plant will parallel the development and maturation of foliage each year. In other plants, such as citrus, in which periodic flushes of foliage occur during a year, the sensitivity of the plant will show periodic changes.

The age of the needle in relation to the occurrence and kind of exposure can influence the distribution of injury (needle tip necrosis) on conifers. In pines, spruce, and fir, for example, needle elongation is relatively synchronous over the entire tree. Therefore, relatively greater homogeneity in the response could be expected when needles on a single shoot (and in the pine's needles within the same fasicle) are compared. On larch, in which needles appear first on the spurs and then on the new shoots, a difference in occurrence or severity of injury may be used as an indication of the time at which the exposure occurred. Discontinuities or heterogeneity in injury to the foliage of western red cedar or hemlock may also be used as an indication of the interaction between stage of development and time of exposure.

Different species of plants have different tolerances for fluoride, and symptoms should be expected to be most frequent and most severe on the least tolerant species. But this would be true only if the foliage of all the species were sensitive at the time that the exposure occurred. Differences in the age of foliage at the time of exposure can produce apparent anomalies, as illustrated by two examples. Consider two species of plants, a coniferous (sensitive) and a deciduous (tolerant) plant; symptoms are present and more severe on the deciduous species because it broke dormancy first, and the exposure occurred before the new shoots had emerged from the conifer. Suppose also that the same condition is observed when the observations are made in late summer. The more tolerant species may have symptoms because of its indeterminate growth: its newer foliage, which is present at the time of exposure late in the season, is more sensitive than the more mature foliage of the conifer. Although it is important to know the sensitivity of the species, it is also important to know their growth characteristics and the way in which exposures to gaseous fluoride may or could have occurred. It cannot be emphasized too strongly that an evaluation of symptoms must consider not only the severity and kind of symptom, but also the distribution of the symptom in relation to the pattern of growth for that species.

Environmental Factors

The edaphic factors that have been found to affect the tolerance of plants to HF are mineral nutrient status, soil temperature, and soil moisture. Nitrogen deficiency increases sensitivity of beans and tomato. Calcium, magnesium, potassium, and phosphorus deficiencies increase the sensitivity of tomato. On the other hand, excess levels of nitrogen, phosphorus, or calcium in the root zone also increase the sensitivity of tomato. In gladiolus, deficiencies of iron or manganese have no effect; while deficiencies of phosphorus and potassium increase, and deficiencies of nitrogen and calcium decrease the severity of tipburn. Field reports indicate that apricots and other fruit trees show greater necrosis in those parts of an orchard where soil moisture is less available. On the other hand, reduced soil moisture availability increases tolerance in the experimental fumigations of beans. The accumulation of fluoride by the aerial portions of some crops decreases as soil temperature increases.

The major climatic factors that affect the tolerance of plants to HF are light, temperature, relative humidity, and wind speed. In general, plants exposed to HF in the dark are more tolerant than those exposed in the light; however, the exact nature of the modifying effect of light depends upon the species. In sensitive species the effect of darkness appears to be an effect on foliar accumulation: less fluoride accumulates in the dark, but injury in light and dark is associated with about the same foliar concentrations of fluoride. In tolerant species less fluoride accumulates in the dark but injury is associated with lower foliar fluoride concentrations than in the light. Moreover, in one species the effect of fluoride is latent: no injury is present after a fumigation in the dark, but injury rapidly appears in the light after the fumigation is stopped. A greater amount of foliar injury on gladiolus occurs when the temperature is increased during exposures. Also, when gladioli are exposed to HF at different relative humidities, a greater amount of injury occurs at 85% than at 50% or 65% relative humidity. Plants exposed to air pollutants tend to be more sensitive or show more injury when the velocity of air past the foliage is greater.

Although several environmental factors are known to affect the tolerance of plants to HF, the effects of others are still to be determined. Environmental factors enter into the diagnosis of injury in three major ways. First, they may produce symptoms that mimic those produced by atmospheric fluoride. Second, in combination with genetic factors they can affect the tolerance of the plant. Third, to the extent that environment alters or determines the growth cycle of the plant, it thereby indirectly affects the plant's sensitivity to HF and the expression of symptoms.

The mimicry of fluoride by environmental conditions can be detected if the conditions and their symptoms are known. Genetically- and environmentally-produced variability can be accounted for in different ways. For example, the occurrence and severity of symptoms should be expected to be much more uniform in a field of hybrid corn (since it is more genetically uniform) than in a planting of Scotch pine. One kind of interactive effect of environment and plant development in symptom expression has already been illustrated, but another kind occurs as a latency in the expression of an effect. If conifers receive low-level and long-term exposures, fluoride may be taken up by the needles without any symptoms appearing. Later in the growing season, when a hot, dry period occurs that may induce moisture stress, symptoms may suddenly appear on the fully elongated needles. On the other hand, symptoms may not develop so quickly on some plants when a period of cool, moist weather follows exposure to atmospheric fluoride.

Injury Threshold Doses

Symptoms induced by atmospheric fluoride on plants become more severe and frequent as the concentration, the length of the exposure period, or the frequency of exposures become greater. It is difficult to say what the thresholds for injury may be, but the lowest concentrations of HF and the shortest duration of exposure, for which investigators have reported injury to be present, can be indicated. These kinds of data are illustrated in Figure 5-2, in which individual species are not represented, but general and approximate categories of tolerance – sensitive, intermediate, and tolerant – are indicated. The positions certain species occupy with respect to these categories may be seen in Table 5-2.

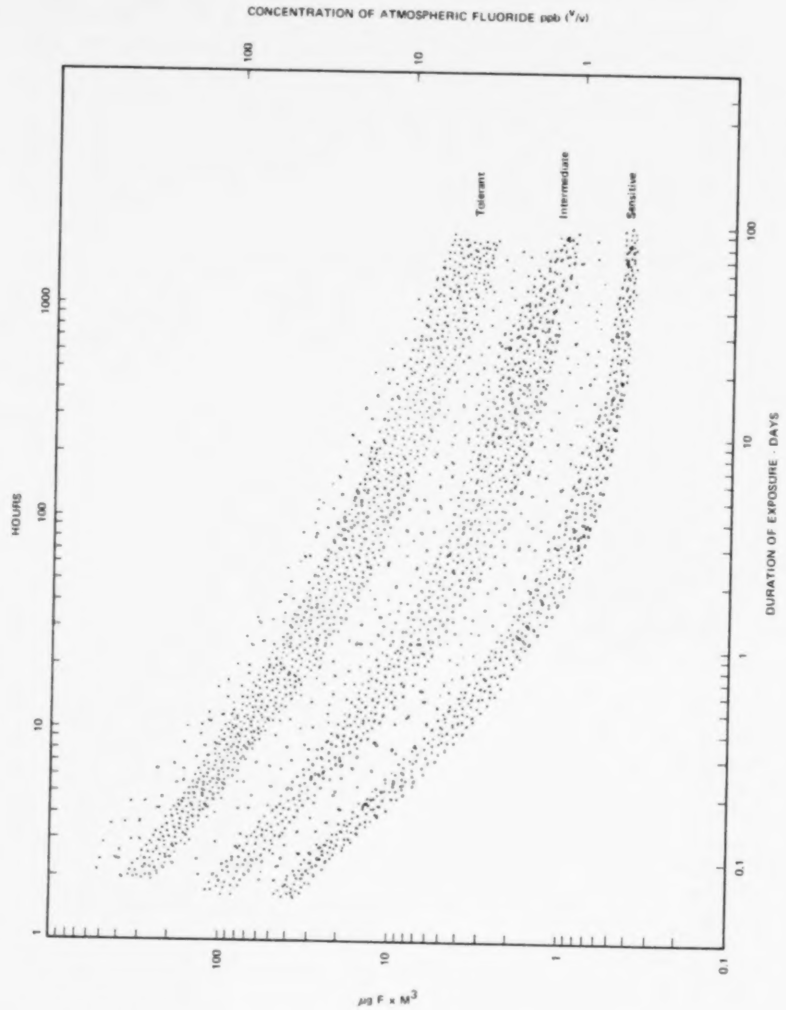

Figure 5-2. *The Relationship of Concentration of Atmospheric Fluoride (Vertical Axis) and Duration of Exposure (Horizontal Axis) to the Threshold for Foliar Symptoms for Sensitive, Intermediate, and Tolerant Species of Plants*

Table 5-2

Relative Sensitivity of Various Plants to F

Flowering Shrubs and Ornamental Plants

Sensitive	*Intermediate*	*Tolerant*
Gladiolus	Azalea	Mock-orange
Jerusalem cherry	Rose	Bridalwreath
Tulip	Narcissus	Firethorn
Iris	Dahlia	Chrysanthemum
	Lilac	Camellia
	Rhododendron	Petunia
	Aster	Privet
	Violet	
	Geranium	
	Peony	
	Sweet William	
	Flowering cherry	
	Flowering plum	
	Jerusalem cherry	

Weeds

Sensitive	*Intermediate*	*Tolerant*
St. John's Wort	Johnson grass	Burdock
Crabgrass	Nettle-leaf goosefoot	Nightshade
Smartweed	Goldenrod	Dock
Chickweed	Sunflower	Plantain
Lamb's quarters		Purslane
Pigweed		
Johnson grass		

Conifers

Sensitive	*Intermediate*	*Tolerant*
Pine, Ponderosa, Scotch (young needles)	Grand fir	Pine, Lodgepole, Mugo (mature needles)
Western larch	White spruce (young needles)	Arborvitae
Douglas fir	Spreading Japanese yew	Juniper
Blue spruce		

Broad-leaved Trees and Shrubs

Sensitive	*Intermediate*	*Tolerant*
Oregon grape	Serviceberry	Sycamore
Boxelder	Quaking aspen	American linden
Serviceberry	Red mulberry	Currant
	Black walnut	London planetree
	English walnut	Sweetgum
	European mountain ash	Birch, white
	Silver maple	Birch, cutleaf
	Common chokecherry	Mountain laurel
		Dogwood

Table 5-2 (Continued)

Relative Sensitivity of Various Plants to F

Broad-leaved Trees and Shrubs (Continued)

Sensitive	*Intermediate*	*Tolerant*
	Poplar, Carolina	Modesto ash
	Poplar, Lombardy	Virginia creeper
	Maple, Norway	Elderberry
	Maple, hedge	Black locust
	Little-leaf linden	Oak
	Sumac, staghorn	Balsam poplar
	Sumac, smooth	Elm, American
	Green ash	Elm, Chinese
		Russian olive
		Tree-of-heaven
		Willow

Fruit Trees

Sensitive	*Intermediate*	*Tolerant*
Italian prune	Apricot, Moorpark	Pear
Apricot, Chinese	Apricot, Tilton	Red raspberry
Apricot, Royal	Peach (foliage)	
Bradshaw plum	Sweet cherry	
Peach (fruit)	Apple	
Blueberry	Lemon	
European grape	Tangerine	
	Grapefruit	
	Orange	
	Concord grape	

Field Crops

Sensitive	*Intermediate*	*Tolerant*
Milo maize	Field corn	Cotton
Barley (young)	Barley (mature)	Tobacco
Oats (young)	Oats (young & mature)	
Rye (young)	Rye (young & mature)	
Wheat (young)	Wheat (young & mature)	
	Crimson clover	
	Sweet clover	
	Soybean	
	Alfalfa	
	Sugar cane	

Garden Crops

Sensitive	*Intermediate*	*Tolerant*
Sweet corn	Tomato	Eggplant
Sweet potato	Strawberry	Asparagus
	Bell pepper	Spinach
	Pea	Celery
	Carrot	Cabbage
	Snap bean	Cauliflower
	Potato	Summer squash
	Eggplant	Cucumber
	Spinach	

Air Quality Standards

No air quality standards have been established for fluorides.

Relative Sensitivity of Plants to HF

Many investigators have compiled lists in which various species are assigned to certain categories on the basis of their sensitivity or tolerance to air-borne fluorides. Different lists sometimes assign the same species to different categories. There are several reasons for this. Most of the rankings are based upon the appearance of symptoms or lesions on leaves. This is quite relevant to the problems of surveying vegetation for the assessment of effects but ignores two other aspects. An effect on productivity or the accumulation of fluoride may be more important than foliar symptoms and is only loosely related to them in a species which is usually classified as tolerant, such as alfalfa. Also, great differences in tolerance exist not only among species but among cultivars of the same species and among individuals in a natural population. Therefore, the category a certain species occupies depends upon what portion of the species was observed. Many rankings are based on field observations whereas others come from experimental fumigations. In some fumigations, a species is rated according to the concentration or the length of exposure that was required to first produce symptoms. In other fumigations, species may be rated according to the degree or severity of injury produced by a single fumigation. There is also the possibility that conditions of experimentation and the kind of response observed may determine the ranking of species.

Leaf Tissue Analysis

As has been mentioned before, the chemical analysis of vegetation for fluoride is an important aspect of field surveys and comprises the following interrelated activities: sampling vegetation in the field; preparing the material for fluoride analysis; and evaluating the analytical results.

Three kinds of problems illustrate how sampling of vegetation is influenced and directed by the particular objective and ways in which environmental conditions, biological factors, and the characteristics of exposure affect accumulation, distribution, and retention of fluoride by foliage.

Aiding or Confirming a Diagnosis

The sampling of foliage to determine if tipburn on a pine is caused by fluoride should be done with an awareness that the fluoride, which may indeed be present, can be both internal and superficial. Dust particles from roads or the soil residing on the foliage may contain fluoride but may not be relevant to that in the tissue or to an injury. If the distinction between these two kinds of fluoride in the sample is wanted, the needles may be partitioned into two sub-samples and one can be washed to remove superficial fluoride before the tissue is dried for analysis. Needles from higher limbs tend to have greater concentrations of fluoride, therefore samples should be taken from at least two elevations on the tree. Since fluoride continues to accumulate in old needles, samples should be taken from current year's needles. Fluoride, after it has been absorbed by the needle, moves to the tip and accumulates. Thus, there should be a difference between tip and basal portions of the needle. However, the translocation of fluoride takes a certain amount of time and if needles are sampled immediately after exposure, the process may not be complete and the difference between tip and base will not be so great. Control samples should be obtained from trees of the same species in an area that is known to be free or remote from a source of atmospheric fluoride. These samples should be treated and analyzed in the same way as those from affected trees so that the results from the affected trees can be compared with a baseline value.

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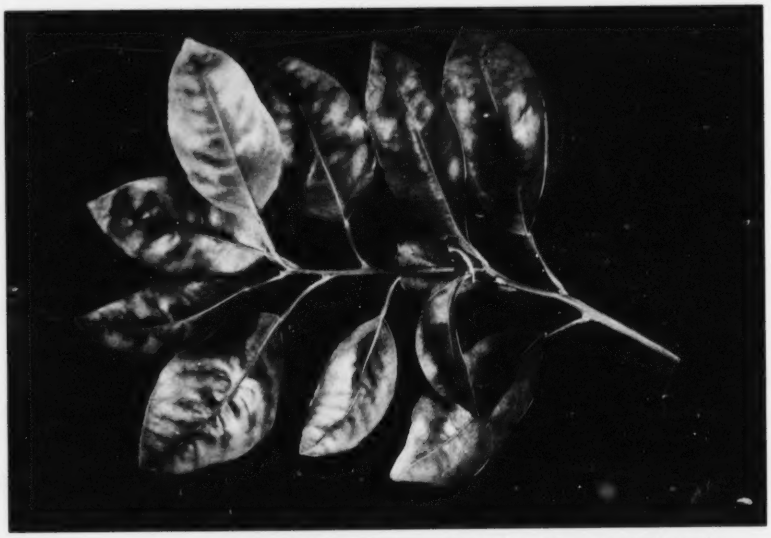


Plate 5-1. *Citrus Foliage with the Typical Fluoride-Induced Pattern of Chlorosis*

Plate 5-2. *Blueberry Leaves Showing the Kind of Injury Induced by Atmospheric Fluoride*

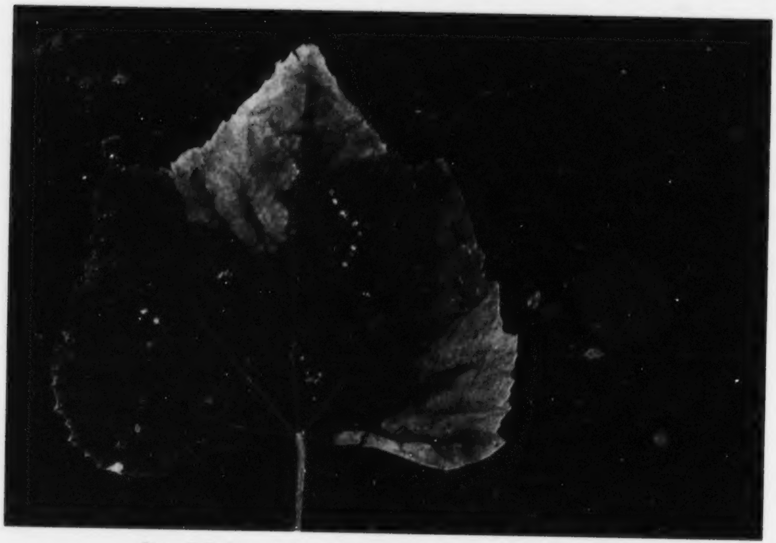


Plate 5-3. *Marginal and Intercostal Necrosis of a Grape Leaf*

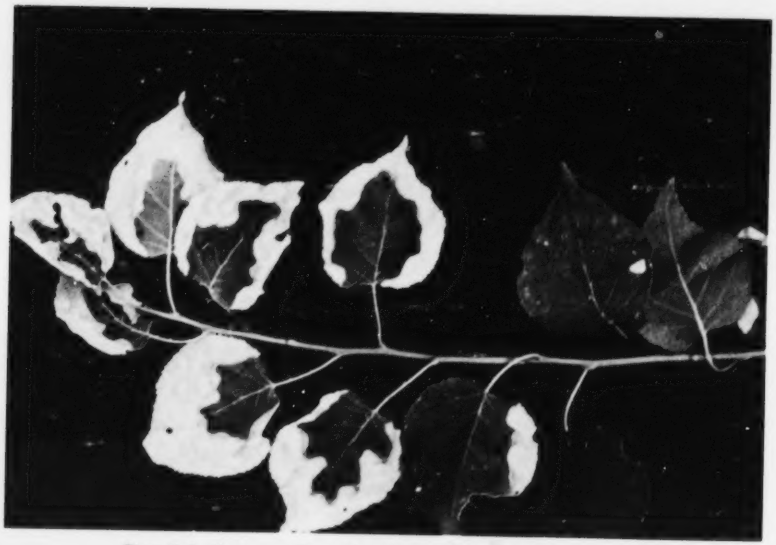


Plate 5-4. *Hydrogen Fluoride Injury to Poplar (Field Exposure)*

Plate 5-5. *Oregon Grape with Symptoms Mimicking Fluoride Injury, Cause Not Known*

Plate 5-6. *Lamb's Quarters Showing HF-Induced Injury*

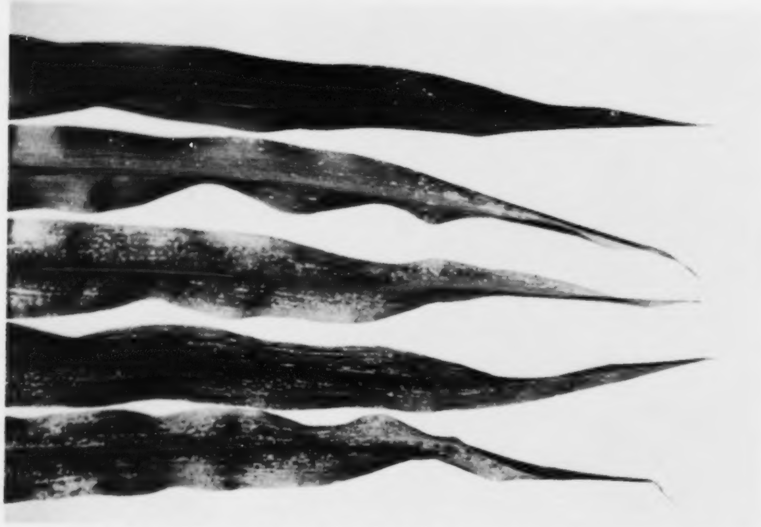


Plate 5-7. *Leaves of Sweet Corn Showing HF Induced Symptoms and Varietal Differences in Tolerance. From Top (Tolerant) to Bottom (Sensitive) the Varieties are: Surecross, Golden Cross Bantam, Gold Rush, Spancross and Marcross*

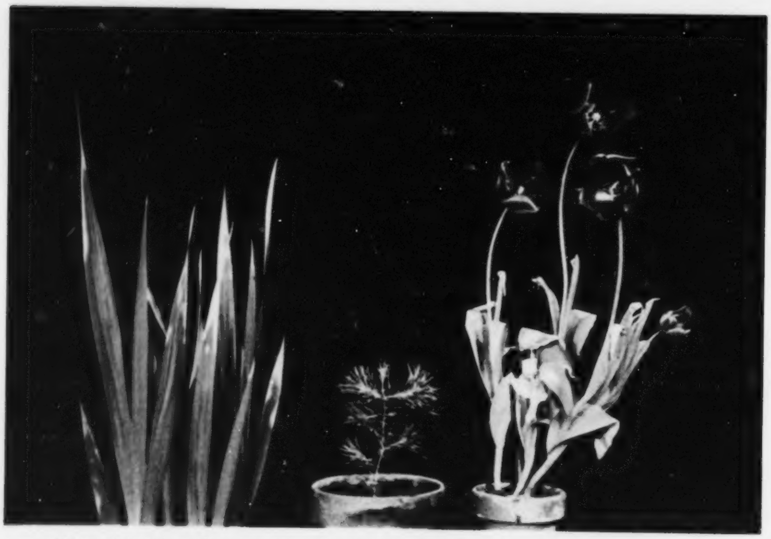


Plate 5-8. *Injury Due to HF on Gladiolus, Pine, and Tulip Fumigated Together*

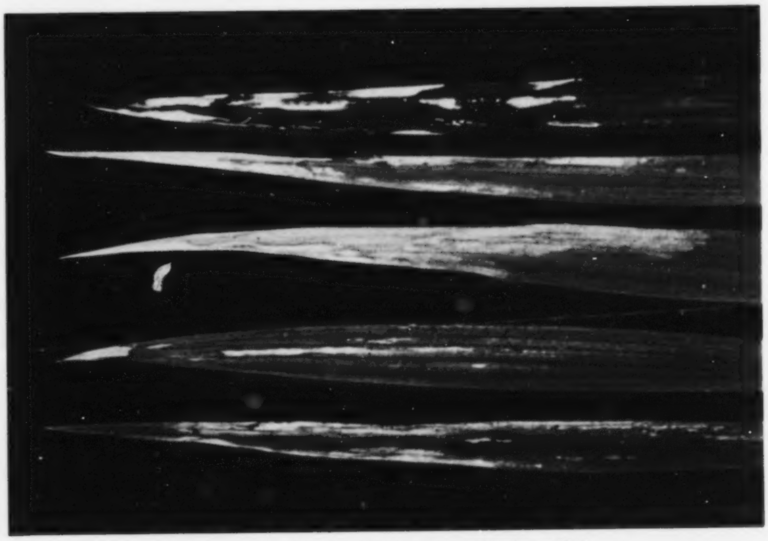


Plate 5-9. *Fluoride-Induced Injury on Gladiolus Foliage*

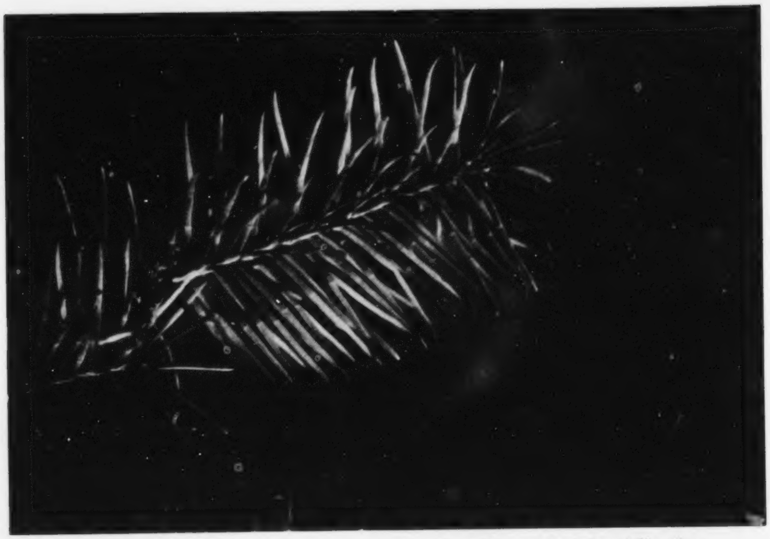


Plate 5-10. *Fluoride-Induced Injury on Fir; Note Several Uninjured Needles*

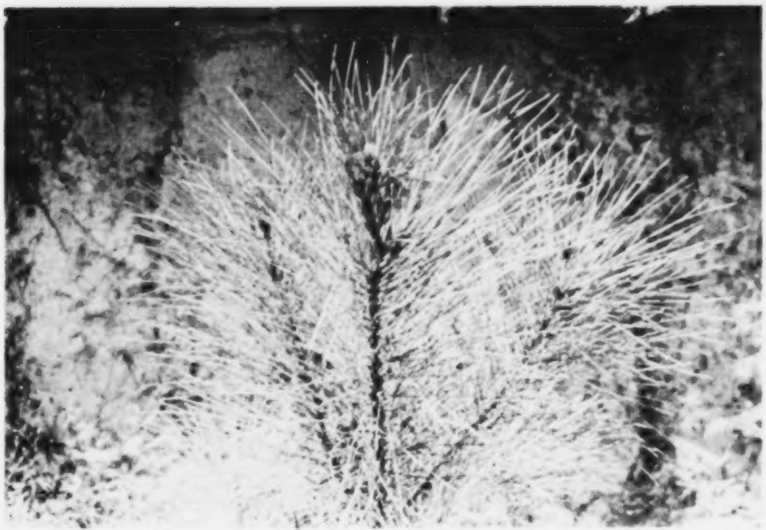


Plate 5-11. *Slash Pine Displaying the Reddish-Brown Discoloration of Needles Injured by Fluoride*

Plate 5-12. *Suture Red Spot Syndrome of Peach Fruit Induced by HF on Dwarf Elberta*

Plate 5-13. *Fluoride Injury to Ponderosa Pine. Note That Only One Tree Exhibits Injury. This is an Illustration of the Natural Differences in Tolerance Found in the Field*

Chapter 6

MINOR POLLUTANTS

Introduction

This chapter contains information on a number of pollutants generally referred to as "minor pollutants" because they have not yet resulted in major social or economic problems. Some of these, such as ethylene, are widely distributed but seldom cause damage because the concentration is usually too low. Others, such as carbon monoxide, do not cause plant damage at concentrations normally found even in polluted atmospheres. Still others are not ubiquitous but occur only as accidental releases. Sources of the pollutants and relative frequence of injury from these pollutants are presented under each pollutant. The same format has been followed in this chapter as in previous chapters.

SECTION 1. CHLORINE

Introduction

Chlorine (Cl_2) is a yellowish-green gas that is widely used commercially because of its strong oxidizing property. Nearly 10 million tons of the gas are produced annually in the U.S. About 70% of this amount is used by the chemical industry in the manufacture of myriads of products from plastics to pesticides. About 18% is used by the pulp and paper industry, and the balance is used for sanitary purposes, such as water and sewage purification.

Sources

There are only 69 industrial plants producing this gas in the U.S. Although there are a number of potential sources of atmospheric emissions in the manufacturing process, the manufacturing process has not been responsible for much vegetation damage. Because chlorine is very hazardous to all forms of life, the manufacturing, handling, and shipping processes are carried out with much precaution. Even when tank cars or cylinders are returned to factories for refill, the containers are evacuated, and this gas is used for products requiring low grade gas. However, since the gas is so widely used, there are a number of potential user sources.

Damage to vegetation by this gas has usually resulted from accidental releases such as breaks in lines or faulty connections in equipment using chlorine. Accidental leaks usually result in very high concentrations of the gas for short periods of time in a limited area. The injury that has been observed in the field has reflected this pattern, i.e., plants near the source are severely damaged or killed, and the severity of symptom expression decreases with distance. Damage to vegetation due to chlorine gas was reported to have occurred over one hundred years ago in Europe near brick and clay product factories and chemical plants.

Natural sources of this gas include that released from sea salt particles in marine air, emissions from volcanic eruptions (about 9 million metric tons annually), crustal weathering, and forest fires.

Atmospheric Chemistry

Much of the chlorine compounds emitted from natural sources is removed from the atmosphere in relatively close proximity to the point of origin by precipitation and dry fall-out. For example, about 90% of the chlorine produced by the oceanic source is returned to the sea, and only 10% is deposited on the continent. Chlorine is a very reactive chemical and will undergo changes very quickly in the atmosphere. Chlorine-containing compounds can exist in gaseous form, or combined with material in aerosols.

Chlorine compounds are dispersed in the atmosphere just as any other trace contaminant. The chlorine compounds may react chemically in the atmosphere and be transformed into materials including oxidized species. The atmospheric reactions of chlorine compounds have not been studied extensively. However, a number of photochemical reactions have been postulated.

Monitoring Data

Only a very few measurements of gaseous chlorine have been taken in uncontaminated atmospheres. In coastal areas the concentration of chlorine is usually about 0.001 ppm (2.9 $\mu g/m^3$). Since chlorine is not as ubiquitous nor as abundant as other pollutants, it is not measured routinely in cities of the U.S. The average concentration of total chlorine in large cities such as Baltimore and Cincinnati is 0.02 ppm (58.0 $\mu g/m^3$). No information is available in the public literature on ambient concentrations near point sources nor on long-term trends.

Symptomatology

Acute Injury

Broad-leaved (Dicotyledonous) Plants. The most characteristic symptom on these plants is a spotting of the leaf. The spots first appear as cooked (very dark) green areas that later turn straw-colored or brown (Plates 6-1, 6-2). These are sometimes located between the veins, but usually they are found close to the margin, and increase with the intensity of the fumigation and progress towards the midrib. In some instances, chlorine injury will result in an upper-leaf surface flecking and necrosis similar to ozone injury (Plate 6-3) or in an interveinal bleaching similar to sulfur dioxide injury (Plate 6-4).

Narrow-leaved (Monocotyledonous) Plants. On grasses exposed to chlorine, the first symptoms to appear are marginal necrotic streaks that progress toward the main vein of the leaf with time. Symptoms usually develop in the region between the tip and the point where the grass leaf bends over.

Coniferous Plants. Tip necrosis (Plate 6-5) has been the most common symptom observed on conifers in artificial fumigations. The necrosis extends toward the base of the needle as the severity of exposure increases. The necrosis may be tan, brown, or reddish in color. Mottling of the needle may also be present (Plate 6-6).

Exposure to chlorine often produces somewhat different symptoms. On some plants, such as mustard, annual and Kentucky bluegrass, chickweed, nettle-leaf goosefoot, and lamb's quarter, white to tan markings may be produced. In some instances, chlorine may produce an overall bleaching without killing the tissue and resulting in a discoloration that may range from pure white to light green or yellow.

Chronic Injury

Very little is known about the chronic effects of chlorine gas. In experimental work that has been reported, epinasty has been reported to occur without foliar markings, and sometimes leaf cupping and abscission may be present.

Mimicking Symptoms

In general, chlorine injury symptoms are similar to those reported for ozone. On conifers, chlorine injury causes a tip necrosis and needle mottle that looks very similar to ozone injury.

Factors Affecting Plant Response

Very little information is available on the factors that affect plant response to chlorine. Plants growing under low soil moisture conditions are less severely injured than those grown under high soil moisture conditions. The age of the plants appears to be important; for example, six-week-old plants are more sensitive than three-week-old plants. However, age interacts with soil moisture, and plants of different ages will react uniformly when growing on dry soil.

The presence of moisture on the leaf does not affect plant sensitivity. Shade has a tendency to minimize damage when the shade period follows fumigation. No effect is noted if the shading precedes fumigation. Water stress, even slight wilting, also causes plants to become tolerant. Hardened vegetation (e.g., pine trees in winter) is less sensitive than actively growing vegetation.

Injury Threshold Doses

Threshold concentrations for acute injury are reported to be in the vicinity of 0.5 to 1.6 ppm (1.4 to 4.35 mg/m^3) for exposures of one-half to three hours as summarized in Table 6-1.

Table 6-1
Threshold Concentrations for Acute Chlorine Injury

Species	*Conc. mg/m^3*	*Time, Hrs.*	*Dose (Conc. x Time)*
Buckwheat	1.6	2	3.2
Peach	1.6	3	4.8
Bean	3.8	0.5	18.0
Radish	3.8	0.5	18.0
Rose	4.4	0.5	22.0

Air Quality Standards

No air quality standard has been established for chlorine in this country. Russia has established a 24-hour average maximum allowable concentration of 0.01 ppm (29 μg/m^3).

Relative Sensitivity of Plants to Chlorine

Several investigators have provided information regarding relative sensitivity of plants to chlorine. Table 6-2 is compiled from the sources listed under *Selected References.*

Table 6-2

Relative Sensitivity of Various Plants to Chlorine

Sensitive	*Intermediate*	*Tolerant*
Alfalfa	Annual blue grass	Azalea
Blackberry	Buckwheat	Balsam fir
Box elder	Cheeseweed	Begonia
Chickweed	Corn	Cactus
Crab apple	Cucumber	Chenopodium
Gomphrena	Dahlia	Chrysanthemum
Horsechestnut	Dandelion	Cowpea
Mustard	Nasturtium	Field corn
Pin oak	Nettle-leaf goosefoot	Geranium
Radish	Onion	Kentucky bluegrass
Rose	Petunia	Lamb's quarter
Sugar maple	Pinto bean	Mignonette
Sunflower	Scotia bean	Myrtle
Sweet gum	Squash	Oxalis
Tobacco	Tomato	Pepper
Tree-of-heaven		Pigweed
Tulip		Pine
Virginia creeper		Polygonum
Wandering Jew		
Zinnia		

Leaf Tissue Analysis

Although several methods are available for detecting chlorine from plant tissue, it has been reported that plants exposed to identical chlorine exposures will give inconsistent results.

SECTION 2. HYDROGEN CHLORIDE

Introduction

Over 2 million tons of hydrogen chloride (HCl) gas is produced annually in the U.S. at about 150 facilities. About 90% of this amount is produced as a by-product in the chlorination of organic compounds. This gas is easily removed from contaminated air streams, and control equipment is available to do so. Up to 99.5% of the gas can be removed from the gas stream. Because the gas is so easily scrubbed by relatively inexpensive and readily available equipment, the manufacturing industry has been quick to put controls on their process when air contamination problems arose. Therefore, very few cases of vegetation damage have been reported near industrial manufacturing plants.

Combustion of fossil and rocket fuels provides another source of HCl gas. For example, an 800 megawatt (MW) power plant burning 0.2% chlorine-containing coal would discharge about 25 tons of HCl gas per day. About 500 pounds of HCl are produced per 100,000 persons per day for domestic space heating. The gas is also given off in the burning of municipal refuse because of the chlorine compounds contained in paper, salt in food, grass clippings, wool, leather, etc. The HCl concentration of the gas stream leaving incinerators is normally about 500 ppm (745 mg/m^3). However, it may be higher in individual incinerators depending on the components of the trash. Vegetation damage was reported near a hospital incinerator where paper comprised 80 to 90% of the trash.

About 25,880 tons of HCl are emitted into the atmosphere each year from the lead chloride additives in motor fuels.

Sources

The effects of hydrogen chloride were first noted in Europe in the vicinity of alkali plants during the mid-nineteenth century. Extensive studies were undertaken at that time and in subsequent years by several German researchers. As a result .of their effort, air scrubbing devices were installed at the offending industries, and the problem was essentially solved. Much of their research however, the descriptive aspects in particular, is of limited value for present purposes because most of the fumigations were performed without adequate air circulation over the plants.

Atmospheric Chemistry

Some investigators have postulated that sulfur trioxide formed by the oxidation of sulfur dioxide may dissolve sea salt particles in marine air, lower the pH, and release HCl. However, since ammonia is also given off by the sea, it appears unlikely that the pH could become low enough to effect the release of HCl.

Hydrogen chloride gas is extremely hygroscopic and probably does not exist as a gas in ambient air following its release. It is probably quickly changed into hydrochloric acid aerosol droplets.

Monitoring Data

There is no information in the public literature on ambient air concentrations of hydrogen chloride.

Symptomatology

Acute Injury

Broad-leaved (Dicotyledonous) Plants. Most commonly, two types of injury occur on broad-leaved plants. Marginal or interveinal necrosis (Plates 6-7 and 6-8) is probably encountered most frequently. The necrotic tissue may become yellowish-brown, brown, reddish, or nearly black in color. Brown-orange discoloration of the leaf margin has also been reported. Severe injury on tomato leaves may result in bronzing or glazing of the leaf surface. Quite frequently, the tissue surrounding the brown necrotic tissue is bleached to a cream or pure white color. The second type of injury commonly seen is a flecking or spotting of the upper leaf surface. The spots may be transparent, reddish-brown, black, or variously pigmented depending on the species involved (Plate 6-9).

Narrow-leaved (Monocotyledonous) Plants. No information is available on monocots.

Coniferous Plants. On conifer needles the only symptom reported has been a reddish-brown tip necrosis (Plate 6-10).

Chronic Injury

The only information available on chronic injury is on broad-leaved plants. Chlorosis is the dominant symptom observed when plants are fumigated repeatedly at very low concentrations. The chlorosis is generally interveinal and occurs most frequently on the younger leaves and leaves intermediate in maturity. Upward curling of the leaf and leaf abscission are also sometimes present.

As with many other pollutants, symptom expression appears to be quite variable among different plant species. Some of this variability is undoubtedly related to the hygroscopicity of the gas. Some of the symptoms that have been observed in the field, particularly leaf spotting, are probably the result of hydrochloric acid injury while other symptoms are more typical of a gas-type injury.

Mimicking Symptoms

Under-leaf surface glazing, which may also be caused by hydrogen chloride, was first noted as a response to PAN. Marginal leaf scorch can be caused by a number of agents such as chlorine, soluble salts, drought or wind injury, fluorides, and sometimes sulfur dioxide.

Factors Affecting Plant Response

Only a limited amount of work has been done on the factors that affect plant response to chlorine exposure. Relative humidity appears to be the dominant factor in symptom expression. There appears to be a threshold above which injury will be quite severe and below which the injury is minimal for a given dose.

Age of tissue appears to be another important factor in symptom expression; for example, chloride can be translocated in the leaf and accumulate in the tip much like fluoride so that these tissues may show the greatest injury.

Injury Threshold

Chronic injury (chlorosis) will develop in tomato when fumigated at 0.7 ppm (1.04 mg/m^3) for ten hours per day for two days. No visible injury results at 0.4 ppm (0.6 mg/m^3) for eight hours per day for six days.

Acute injury is more variable. About 4 ppm (6.0 mg/m^3) for two to four hours is required to injure sensitive broad-leaved plants at relative humidities conducive to injury. Up to 10 ppm (14.9 mg/m^3) for the same time periods is required to injure those same species at low relative humidity (less than 50%).

Tolerant broad-leaved plants, such as chrysanthemum, are injured at concentrations between 4 and 9 ppm (6.0 to 13.4 mg/m^3) for three hours.

Conifers are injured at concentrations starting at 8 ppm (11.9 mg/m^3). Some hardwoods (e.g., tulip poplar) are sensitive at 3 ppm (4.5 mg/m^3) for four hours, while others are tolerant even at 13 ppm (19.4 mg/m^3) for four hours.

Air Quality Standards

No standard has been set for hydrogen chloride in the U.S., but Russia has established 0.009 ppm (13 μg/m^3) as a 24-hour maximum average.

Relative Sensitivity of Plants to Hydrogen Chloride

Investigators have provided information regarding relative sensitivity of plants to hydrogen chloride. Table 6-3 is compiled from the sources listed under *Selected References.*

Table 6-3

Relative Sensitivity of Various Plants to Hydrogen Chloride

Sensitive	*Intermediate*	*Tolerant*
Caespitose phlox	Black cherry	Adonis
Columbine	Chrysanthemum	Arborvitae
Cornflowers	European black alder	Austrian pine
Garden daisies	Norway maple	Balsam fir
Oriental poppy	Sugar maple	Douglas fir
Tomato	Pinto bean	Garden iris
Tulip tree	White pine	Garden lupin
		Garden peony
		Goldenrod
		Larkspur
		Lily-of-the-valley
		Norway spruce
		Paniculate phlox
		Plantain lily
		Pheasant's eye pink
		Red oak
		Solidago
		Sweet William

Leaf Tissue Analysis

Good results have been reported for detecting chloride in plants fumigated with hydrogen chloride. Several methods are available, and one should consult chemistry references for method of choice and approved methods.

SECTION 3. ETHYLENE

Introduction

Of all the hydrocarbons emitted into the atmosphere by both natural and technological sources, ethylene appears to be the only one that poses a direct threat as an air pollutant to vegetation. Its effects on plant life were first observed well over a hundred years ago when city trees were killed by illuminating gas leaking from underground mains. It was not until the turn of the century, however, that ethylene was shown to be the causative agent of injury in illuminating gas. Paradoxically, this gas (in the form of kerosene smoke from space heaters) was also being used commercially by the horticultural industry at that time to ripen fruit. Subsequent research showed that ethylene is produced by plants and is also an important growth-regulating hormone. The importance of ethylene as an air pollutant at the present time, however, bears no relationship to its original source as a phytotoxicant.

Sources

Ethylene is a colorless gas with two carbon atoms joined by a double bond and which smells somewhat like ether. The natural sources of the gas are principally production by plants (estimated at 20,000 tons annually in the U.S.) and soils, volcanic eruptions, forest and other wild fires, and, to a minor extent, leaking natural gas. However, the greatest contribution to the atmospheric burden comes from the activities of man. Automobile emissions provide the greatest volume, but other motorized transportation vehicles, stationary fuel combustion units, various industrial processes such as brick kilns, foundries, coke ovens, gas works, polyethylene plants, and miscellaneous sources such as refuse and agricultural burning also contribute. It has been estimated that nearly 60,000 tons of ethylene are released to the atmosphere annually in the U.S. as a result of open burning.

Research on the phytotoxicity of ethylene in comparison to other pollutants has been extremely limited. It could be concluded that the low level of activity is simply a reflection of its relative economic and academic significance. Although most of the economic losses due to this pollutant have been limited to commercial flower growers, it is possible that considerably more injury is occurring but is not detected or reported. It could likewise be concluded that the lack of research on ethylene as a phytotoxic air pollutant precludes an accurate evaluation of its relative importance among other pollutants. Future research, however, may reveal subtle effects not readily noticed by field observation.

Atmospheric Chemistry

Ethylene is continuously emitted into the atmosphere by a number of sources, and has upon occasion accumulated in the air to levels injurious to plant life. Ethylene is known to react with other atmospheric pollutants such as ozone and oxides of nitrogen. With ozone, ethylene may form both carbon monoxide and carbon dioxide, formaldehyde, water, and formic acid. Ethylene also reacts with nitrogen oxide and nitrogen dioxide in the presence of sunlight to form ozone and formaldehyde.

Soil microbes are able to consume hydrocarbons, including ethylene. Green plants, however, do not remove the gas from the atmosphere, although there are reports of uptake by fruit.

Monitoring Data

Because hydrocarbons are important in the formation of photochemical smog, they are routinely monitored by regulatory agencies. However, only olefin hydrocarbons are reactive. Furthermore, since the olefins are all involved in the photochemical reactions, it is not important to separate and measure each one in atmospheric analyses. Most instruments in use at the present time measure the hydrocarbon methane (CH_4), which is nonreactive, and total hydrocarbons. The difference between the two readings is the fraction of interest in terms of photochemical smog. Unfortunately, this reading gives no indication of the amount of ethylene present in the sample. Consequently, only very few data are available on ethylene concentrations in ambient air.

It has been reported that ethylene is normally absent or present only in small amounts in rural air, less than 5 ppb (5.75 $\mu g/m^3$). A number of researchers have reported concentrations in large cities in excess of 100 ppb (115 $\mu g/m^3$). In the Washington, D.C. area in July and August of 1972, the average ethylene concentration ranged from 700 ppb (805 $\mu g/m^3$) downtown, to 39 ppb (44.8 $\mu g/m^3$) outside the beltway. In Denver, measurements made during October, December, and February 1972-73, indicated an average concentration of 37 to 45 ppb (42.6 to 51.8 $\mu g/m^3$).

Symptomatology

As a natural plant hormone ethylene plays a role in abscission, regulation of growth, and other physiological functions. Since these are normal functions, they will not be discussed *per se* in this section. Two types of symptoms are of interest here: Those resulting from traumatically-induced ethylene production, and those resulting from exposure to elevated concentrations of ethylene in the ambient atmosphere. Although there is overlap, and the distinction may be artificial, they will be discussed separately for convenience.

Traumatically-Induced Ethylene

Ethylene production in plants is stimulated by insect infestations, temperature extremes, water stress, diseases, mechanical injury, ozone injury, chemical injury, and other stresses. The symptoms produced by such stresses include senescense and abscission of plant parts. One well-known example is the abscission of cotton bolls following infestations by the cotton boll weevil. Accelerated or premature ripening is another symptom associated with increased internal ethylene. The practice of mechanically injuring figs to stimulate fruit development and ripening dates back to the Egyptian civilization.

A number of chemicals are known to increase ethylene production through hormonal or phytotoxic reactions. Some of the chemicals, such as the herbicide

Endothal, are used commercially as defoliants. Recently, ozone has been demonstrated to stimulate ethylene production in certain plants. This may explain why plants exposed to low concentrations of ozone exhibit typical ethylene symptoms, namely, epinasty, abscission, and premature senescence.

Exposure to Ambient Air

Acute Effects. The best known acute symptom caused by ethylene is "dry sepal" of orchid (Plate 6-11). This disorder is characterized by a drying and bleaching of the sepals starting at the apex and progressing toward the base. The concentrations required to produce this symptom are 0.04 to 0.1 ppm (4 to 115 $\mu g/m^3$) for eight hours, or 0.002 to 0.02 ppm (2.3 to 23 $\mu g/m^3$) for 24 hours.

There have been many reports of damage to trees, shrubs, and grasses from leaking gas mains. This problem was particularly troublesome around the turn of the century when illuminating gas was in widespread use. The ethylene component in illuminating gas was shown to be the principal cause of damage. The gas in use at the present time is natural gas and contains no ethylene. Damage to vegetation from leaking natural gas is presently thought to be the result of roots drying out and lack of oxygen available to roots because the gas fills the pore spaces in the soil.

Chronic Effects. A number of symptoms, mostly involving growth abnormalities, are associated with exposure to ethylene. These include epinasty, abscission (Plate 6-12), hypertrophy, premature bud break, inhibition of leaf expansion, and leaf curling. Perhaps the best known and most frequently observed symptom is that on flowering. Actually, three separate effects have been described: inhibition of flowering, promotion of flowering, and sex reversal. Promotion of flowering occurs primarily with bromiliads (e.g., pineapples) but concentrations much greater than would be encountered in smoggy air are necessary to induce this symptom. A six-hour exposure to 100 ppm (115 mg/m^3) is necessary to induce flowering in pineapples. However, such elevated concentrations could occur in a closed greenhouse with faulty space heaters, for example. Sex reversal occurs with cucurbits. Horticulturists have capitalized on this phenomenon by treating plants with ethylene derivatives to increase the number of female flowers. Again, the concentration required to induce this symptom is similar to that required for promotion of flowering.

Inhibition of flowering, commonly called "sleepiness" or "bullheading," has been observed on many species of flowering plants. It was first described on carnation, in which 0.5 ppm (575 $\mu g/m^3$) of ethylene for 12 hours caused opened blossoms to close, and 1 ppm (1150 $\mu g/m^3$) for three days prevented blossoms from opening. However, concentrations as low as 0.03 to 0.06 ppm (34.5 to 69.0 $\mu g/m^3$) for 48 hours can be injurious. Differences in sensitivity have also been noted (Table 6-4).

Loss of apical dominance, or prostrate growth of normally upright plants, has also been reported. Although these symptoms are not deleterious to plants, they may result in economic losses to commercial growers.

The chronic symptoms described above, in one sense, represent "typical" symptoms. However, many of these symptoms are characteristic of natural

Table 6-4

Dose Response of Plants to Ethylene

Response	*$\mu g/m^3$*	*ppm*	*Time*
Abscission			
Cotton squares	46-3,435	0.04-3.0	??
Leaves	345-685	0.3-0.6	5-30 days
Buds	115	0.1	8 hrs.
Petals	575	0.5	1 hr.
Chlorosis (of leaves)	685-2,290	0.6-2.0*	1-30 days*
Dry Sepal (orchids)	5.75-115	0.005-0.1*	8-24 hrs.*
Epinasty	1.15-3,435	0.001-3.0*	3-20 hrs.*
"Sleepiness"	115-1,145	0.1-1.0*	20 hrs.-3 days*
Growth Inhibition	685-3,435	0.6-3.0*	up to 30 days*
Loss of Apical Dominance	46-3,435	0.04-3.0*	??

*The lower concentration for the longer time period produces a similar effect to the higher concentration for shorter time periods.

Source:
Adapted from: *Air Quality Criteria for Hydrocarbons.* Washington, D.C.: Public Health Service, Department of Health, Education, and Welfare, 6-6.

senescence and environmental stresses such as water stress, bacterial wilts, and root rots. Consequently, ethylene injury could easily be confused with other disorders. An interesting example of ethylene injury occurred in a cotton field in Texas in 1957. Ethylene emissions from a polyethylene manufacturing plant located near cotton fields caused considerable damage to the crop. Abscission of cotton squares on plants within one mile of the plant was severe, and loss of apical dominance resulting in prostrate growth was extensive.

Some researchers have suggested using orchid, carnation, snapdragon, cowpea, or cotton as indicator plants to detect the degree of ethylene pollution. The epinastic response of peas has been widely used in laboratory experiments to make rough measurements of ethylene concentrations. The "receptivity" of the plant, however, is important in this respect, as will be discussed below.

Factors Affecting Plant Response

Physiological age appears to be important in the epinastic response of leaf petioles to ethylene. Only young leaves are affected and will bend. The ability of this gas to cause curvature is also dependent upon a supply of auxin from the leaf.

Air Quality Standards

There is no air quality standard for ethylene *per se.* Ethylene is but one of many hydrocarbons that participate in the photochemical reactions of the atmosphere. Rather than singling out ethylene, the regulatory agencies have chosen rather to set a collective standard for all non-methane hydrocarbons. The standard is set at a concentration of 0.3 ppm (200 $\mu g/m^3$) during the hours of 6 to 9 a.m. Interestingly, this standard was not established at that level to preclude detrimental health or property damage effects by hydrocarbons, but rather because monitoring data indicated that if non-methane hydrocarbons were kept below that level, injurious levels of oxidants would not be reached later during the day.

Until more monitoring data are gathered specifically on ethylene, it is impossible to state whether this standard is adequate. Losses of considerable economic consequence have occurred, particularly in the commercial greenhouse-flower industry. However, because of the lack of specific monitoring equipment, such losses have been difficult to document.

Relative Sensitivity of Plants to Ethylene

As with other pollutants, the duration of exposure must be considered together with the concentration in determining the sensitivity and impact. This represents the dose, and provides the best measure of sensitivity (Table 6-5).

Table 6-5
Relative Sensitivity of Various Plants to Ethylene*

Sensitive	*Intermediate*	*Tolerant*
African marigold	Pepper	Dahlia
Orchid	Tomato	China aster
	Rose	Forget-me-not
	Snapdragon	Lobelia
	Cowpea	Sweet pea
	Carnation	Viola
	Lily	Acacia
	Cotton	Calendula
	Ageratum	
	Larkspur	
	Zinnia	

*This table is an arbitrary classification based on available data.

Leaf Tissue Analysis

Since ethylene is produced internally by the plant, it would be impossible to determine what fraction was from an external source and what fraction from the internal source.

SECTION 4. AMMONIA

Introduction

Injury to vegetation from ammonia is rare, and has usually resulted from accidental spills rather than continuous emissions to the atmosphere. Most of the ammonia, about 85%, produced in this country is consumed by the agricultural industry as fertilizer either as anhydrous ammonia or fertilizers derived from ammonia. It is also used widely in the synthesis of organic compounds such as drugs, plastics, and dyes. Recent occurrences of plant damage from ammonia resulted from spills in the transport of the gas and from breaks in pipe lines. Increased usage of anhydrous ammonia as a fertilizer may well result in increased local incidences of damage. However, since it is a commodity of economic significance, and since methods to control industrial losses are readily available, it will probably not become a widespread problem.

Sources

The major portion of the ammonia present in the ambient air is derived from biological processes both on land and in the oceans. These sources include decomposition of organic matter, such as in the composting process in mushroom production, and from animal wastes, such as near large feedlots. Ammonia is also produced in the combustion of fossil fuels; however, no reports of damage from this source have ever been noted.

Atmospheric Chemistry

Ammonia gas is very hygroscopic and probably combines with water to form ammonium hydroxide very quickly once it enters a humid atmosphere. No information is available, however, on the fate of ammonia gas released into the air as a pollutant. Ammonia released from biological decomposition is brought back to the earth by rain, where it is reused by plants. Presumably, the same phenomenon would occur for the ammonia entering the atmosphere from industrial releases. If SO_2 is present in the atmosphere, reactions occur rapidly with ammonia to form ammonium sulfate particles which tend to settle out in time.

Monitoring Data

The National Air Surveillance Network made measurements of ammonia in various cities in the U.S. during 1963 and 1964. There is a background concentration in the lower troposphere of 0.009 ppm (6 $\mu g/m^3$) in temperate latitudes, and 0.2 ppm (140 $\mu g/m^3$) near the equator. The concentration in cities across the U.S. varies between .00014 ppm (0.1 $\mu g/m^3$) and 0.018 ppm (12.7 $\mu g/m^3$). The average concentration for urban areas is 0.029 ppm (20 $\mu g/m^3$). The highest concentration reported was 0.109 ppm (75.5 $\mu g/m^3$) in Ashland, Kentucky. Since ammonia is not routinely measured by regulatory agencies, it is not possible to determine if there have been any trends in atmospheric concentrations.

Symptomatology

A very limited amount of work has been done on the effects of ammonia on plants and consequently the response to this gas has been described for only a few species. The concentrations involved in accidental spills are so great as to render symptom expression on affected plants meaningless. Two interesting observations have emerged, however. The injury caused by ammonia differs from that caused by the more common gaseous pollutants. With ammonia, practically all areas of the leaf are affected rather than a specific area as is common with other gases. Secondly, under certain conditions ammonia increases the pH of the leaf tissue and induces a color change in the leaf.

Acute Injury

This type of injury is characterized by a collapse of tissue without the loss of chlorophyll. The collapsed tissue appears in spots either at the margin of the leaf or, occasionally, in the center. The chlorophyll takes on a dull, dark-green appearance, and, with time, the tissues die and turn brown or black.

On grasses, the injury first develops as an area of very small spots where the leaf bends, giving the leaf a powdery, speckled appearance.

On broad-leaved plants such as sunflower, cheeseweed, pigweed, poison ivy (Plate 6-13), and dandelion, ammonia causes a general browning of the leaf.

Ammonia has been reported to cause glazing and silvering on the lower surfaces of the leaves of vegetable crops.

Chronic Injury

Internal pH changes in the leaf and changes in pigmentation have also been reported which could be considered a chronic response. Generally, though, too little is known regarding low-level, long-term responses to consider chronic effects.

Atypical Symptoms

Fruits, such as peach and apple, may be injured by ammonia. A purple to black discoloration occurs around the lenticels of apples when experimentally fumigated with 400 ppm (278 mg/m^3) of ammonia. Peaches are blackened when exposed to such massive concentrations.

Other symptoms include the response of cotton, mustard, lamb's quarter, annual bluegrass, and Kentucky bluegrass. With these species the leaf markings are white to tan in controlled fumigations (Plate 6-14).

Since ambient ammonia as an air pollutant has not been a generalized problem, no attention has been given to using plants as natural indicators of the presence of this gas.

Factors Affecting Plant Response

Very little has been done in the study of environmental variables that might alter the responses of plants to ammonia exposure. Leaf age is known to be an important factor. Middle-aged leaves, those that have been at full size for a week or more, are the most sensitive. The youngest leaves very rarely show any markings. Soil moisture also modified plant response in that sensitivity increases with increasing moisture.

Injury Threshold

Tomato plants exposed to experimental fumigations of 1,008 ppm (700 mg/m^3) for three minutes undergo changes in the pH of the leaf and stem tissue. This appears to be the lowest concentration at which injury will occur when exposed for such a short period of time. Concentrations of 55 ppm (38 mg/m^3) require one hour to injure plants such as buckwheat, coleus, sunflower, and tomato (Table 6-6).

Air Quality Standards

No standard has been adopted by regulatory agencies in the United States. Czechoslovakia, however, has adopted a 24-hour maximum allowable average of 0.14 ppm (100 μg/m^3); Russia, a 24-hour average of 0.28 ppm (200 μg/m^3); and Ontario, Canada, a 30-minute average of 5 ppm (3,500 μg/m^3).

Relative Sensitivity of Plants to Ammonia

Investigators have provided information regarding relative sensitivity of plants to ammonia. Table 6-6 is compiled from the sources listed under *Selected References.*

Table 6-6

Relative Sensitivity of Various Plants to Ammonia*

*Approximate dose required to cause an effect***		
8.0	*33.6*	*46.0*
Mustard	Kentucky bluegrass	Buckwheat
Sunflower	Dandelion	Coleus
Lamb's quarter	Chickweed	Tomato
Cheeseweed	Nettie-leaf goosefoot	
Annual bluegrass		
Pigweed		

*This table is an arbitrary classification based on available data.
**Dose = mg x time (hours)

Leaf Tissue Analysis

There have been no reports on methods for analyzing plants' tissues for ammonia contamination.

SECTION 5. HYDROGEN SULFIDE

Introduction

Hydrogen sulfide is non-toxic to vegetation at concentrations encountered even in very polluted atmospheres. The concentration required to injure even the most sensitive plants is far above that which is objectionable to humans.

Hydrogen sulfide is emitted from a number of technological activities. Perhaps the most familiar is kraft paper mills. About 64,000 tons of the gas were emitted from kraft mills in 1960 on a worldwide basis. Other anthropogenic sources include the petroleum industry, coking operations, burning culm piles, foundries, and certain chemical industries such as the manufacturers of sulfur dyes, viscose rayon, certain pesticides, and many other organic sulfur chemicals. A number of inorganic processes, such as the smelting and refining of zinc evolve hydrogen sulfide. It is also generated by animal processing plants in the decomposition of sulfur-containing proteinaceous materials.

Sources

Natural sources of hydrogen sulfide have been estimated to yield between 100 and nearly 300 million tons annually on a worldwide basis. Hydrogen sulfide is released primarily from bacterial decomposition of sulfur-containing proteinaceous material.

The importance of this gas in relation to plant damage is inconsequential compared to other air pollutants. A brief description of its effects is presented for academic interest.

Atmospheric Chemistry

Hydrogen sulfide is relatively stable and does not react photochemically in the atmosphere. However, since it can be oxidized chemically in the sewage treatment process, it is reasonable to assume that some oxidation might occur in an oxidant-laden atmosphere. It reacts readily with paints containing metal salts and with certain metallic surfaces.

Monitoring Data

Hydrogen sulfide is not measured routinely by the National Air Sampling Network. Most cities have average concentrations ranging from 0.0007 to 0.004 ppm (1 to 6 $\mu g/m^3$). Industrial areas may average up to 0.066 ppm (92 $\mu g/m^3$). Background concentrations have been estimated to be between 0.0001 and 0.0003 ppm (0.15 to 0.46 $\mu g/m^3$). No data are available on long-term trends.

Symptomatology

Leaf markings resulting from exposure to elevated hydrogen sulfide concentrations are quite different from those caused by other phytotoxicants. The youngest leaves always show the greatest amount of injury, and the growing points may also be killed. Less than fifty plant species have ever been fumigated under laboratory conditions. Knowledge about the effects of this pollutant is limited at best.

Acute Injury

Broad-leaved plants develop an interveinal white to tan discoloration. Wilting without the typical discoloration has also been reported to occur, and this is followed by scorching of the young tissue. Grasses exposed to this gas develop a powdery appearance between the tip and the bend of the leaf.

Chronic Injury

Chronic fumigations with hydrogen sulfide have not been performed. Sunflowers fumigated with this gas will develop an orange-brown cast on leaves in the bud stage, rather than the usual white to tan chlorosis typical of broad-leaved plants.

Factors Affecting Plant Response

Tissue age appears to be the most important factor in plant response, and, unlike the situation with other pollutants, the growing points of stems and leaves are the most sensitive. Also unlike response to other pollutants, drought or moisture stress seems to increase the sensitivity of plants to very high concentrations, 1,000 ppm (1,390 mg/m^3), of the gas. At lower concentrations, 200 ppm (278 mg/m^3), however, high soil moisture increases sensitivity.

Injury Threshold

Most of the species that have been tested have not been injured at concentrations less than 86 ppm (120 mg/m^3) in five-hour fumigations. Species such as Boston fern, apple, cherry, peach and coleus will even withstand concentrations up to 860 ppm (1,200 mg/m^3). Some species, including buckwheat, castor bean, gladiolus, rose, sunflower, tobacco, cucumber, and tomato have been injured at concentrations between these two extremes (Table 6-7).

Air Quality Standards

No standard has been established for this gas. The odor threshold is between 0.0007 to 0.03 ppm (1 to 42 μg/m^3). The odor becomes offensive at around 7.2 ppm (10,000 μg/m^3).

Relative Sensitivity of Plants to Hydrogen Sulfide

Investigators have provided information regarding relative sensitivity of plants to hydrogen sulfide. Table 6-7 is compiled from the sources listed under *Selected References.*

Leaf Tissue Analysis

Since sulfur is metabolized by plants, it is possible in some cases to detect increased sulfur content due to contamination. It would not be possible to distinguish which form of sulfur the plants had been exposed to. If other sulfur sources were present, these would most likely contribute to the body burden.

Table 6-7
Relative Sensitivity of Various Plants to Hydrogen Sulfide

*Minimum dose at which 50% leaf injury will occur**	
600	*3,000*
Lamb's quarter	Chickweed
Nettle-leaf goosefoot	Dandelion
	Sunflower
	Kentucky bluegrass
	Pitweed
	Annual bluegrass
	Mustard
	Cheeseweed

*Dose = mg/m^3 × time (hours).

SECTION 6. CARBON MONOXIDE

Introduction

Like hydrogen sulfide, carbon monoxide (CO) is not toxic to vegetation at concentrations encountered in polluted atmospheres. However, it is the most widely distributed and the most commonly occurring air pollutant. Total emissions exceed those of all other pollutants combined. However, it is important from the standpoint of human health. The general public is usually interested in this pollutant and, therefore, even plant biologists should be aware of a few facts concerning it.

Sources

Transportation activities represent the largest anthropogenic source. Other sources in this category include any activity in which combustion takes place, such as steam generating facilities, industrial processes, and combustion of solid waste. Interestingly enough, man's contribution of carbon monoxide to the atmosphere accounts for only about 10% of total emissions. The remainder comes from natural sources. These sources consist of emissions from volcanoes, natural gas in coal mines, electrical storms, and forest fires. Carbon monoxide is also formed by certain brown algae (kelps). Concentrations of up to 786 ppm (904 mg/m^3) have been measured above floats of kelps, colonies of marine jellyfish, and other ocean-dwelling organisms.

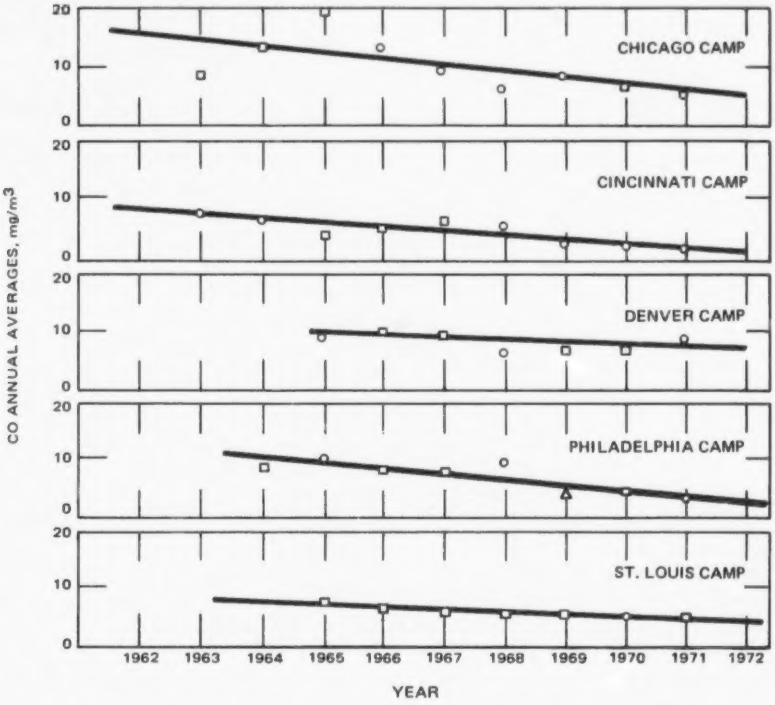
The estimated worldwide emission of CO from anthropogenic sources is 198 to 231 million tons per year. The amount theoretically should have raised the atmosphere background concentration. That has not occurred, however, and a sink or removal process must exist in nature. A few years ago researchers at the Stanford Research Institute discovered that soil microorganisms are capable of removing CO from the soil atmosphere. Fungi, such as certain species of *Penicillium, Aspergillus, Mucor,* and others are particularly efficient at removing this gas.

The trend in carbon monoxide concentrations in large cities, including Chicago, Cincinnati, Denver, Philadelphia, and St. Louis, is encouraging. All of these cities have experienced a decrease in annual average CO concentrations between the period from 1962-66 to 1967-71. This percentage decrease ranges from 18% for Denver to 46% for Chicago. The average percentage decrease was 31%. A summary of annual averages for these cities is presented in Figure 6-1.

Symptomatology

Carbon monoxide has not been found to produce detrimental effects on plants at concentrations below 100 ppm during exposures ranging from one to three weeks. Since the standards have been established at 35 ppm (40 mg/m^3) for eight hours and 9 ppm (10 mg/m^3) for one hour, it appears that vegetation will be amply protected.

When plants are fumigated at concentrations that will elicit responses, the resulting injury has been similar to that caused by ethylene. Epinasty is produced at 500 ppm (575 mg/m^3), while chlorosis and abscission will develop on sensitive plants at 10,000 ppm (11,500 mg/m^3). Tolerant plants can withstand ten times this concentration.



O INDICATES INVALID AVERAGE
(AVERAGE BASED ON INCOMPLETE DATA)

Figure 6-1. *Trend Lines for CO Annual Averages in Five CAMP Cities*

Source: *Air Quality and Emissions Trends Annual Report: Volume I.* Research Triangle Park, North Carolina: National Air Monitoring Program, Environmental Protection Agency, 4-25, 1973.

SECTION 7. HEAVY METALS

Introduction

Heavy metals, such as lead, cadmium, zinc, copper, and mercury, represent a group of pollutants that are currently receiving a good deal of attention. The pollution potential of some of these is relatively unknown while for some others considerable information has already been assembled and is available in review articles. One outstanding characteristic of all members of this class of pollutants is their longevity and stability once they reach the soil. The metals tend to build up in the upper soil layers and are not degraded or leached out with time. If the source of the pollutant persists long enough in an area, the metal(s) accumulate to levels which, in come cases, become toxic to plants or a hazard to other forms of life.

Lead

Lead concentrations have been measured in chronological layers of snow strata in quiescent ice sheets of the frozen polar regions. Annual ice layers from the interior of northern Greenland show that lead concentrations increased from less than 0.005 μg/kg of ice in 800 B.C. to more than 0.2 μg/kg in 1965 A.D. Lead started to increase in 1750 at the beginning of the industrial revolution, and the lead concentration at that time was 25 times greater than natural levels. During the second half of the 18th century, lead concentrations tripled, and from 1935 to 1965, they abruptly tripled again. The sharpest rise occurred after 1940. Today, lead concentrations in Greenland snows are about 400 times earlier levels.

There is evidence also that lead is increasing in other ecosystems. Surface waters in some areas of the Mediterranean and the Pacific have ten times more lead now than in pre-industrial times. The soil in rural areas of the U.S. has a lead concentration usually similar to the average content in the earth's crust, i.e., 10 to 15 μg/g. However, in many cities the concentration of lead in soil and street dust is much higher. The concentration of lead in street dust and surface soil of large cities in the Midwest averaged 1,636 μg/g for residential sites and 2,413 μg/g for commercial sites.

The inorganic emission of lead from the combustion of leaded gasoline constitutes approximately 98% of the total lead emission, or approximately 181,000 tons annually. The concentration of lead in ambient air is closely correlated with the density of vehicular traffic in the U.S. The concentration is largest in cities, smaller in suburbs, and smaller yet in rural areas. There is evidence of diurnal and seasonal cycles. Numerous studies have shown that the concentration of lead in depth profiles of soils along highways decreases with distance from the highway and with depth.

Effects on Plants. Lead does not cause visible injury to plant life. However, plants can become contaminated by lead from three sources: lead in soil, lead in air, and lead present in rainwater or groundwater. Usually, only trace amounts of lead are absorbed by plants from soils, but this amount can be increased by increasing the lead content of the soil or by decreasing the binding capacity of the soil for lead. Some crops, such as lettuce and potatoes, will not translocate lead readily, and lead accumulates in the smaller feeder roots. It appears, however, that in spite of the widely varied concentration of lead in soils, the net effect on the lead content of the plant is negligible because the lead is largely unavailable to the plant. Lead in rainwater also does not appear to cause any detectable change in the lead content in any part of crops.

Several studies have shown that lead in air increases the concentration of lead on the leafy parts of plants near highways. All of the studies seem to indicate that lead accumulates mostly *on* the foliage. About 50% of the lead that is accumulated on plants can be washed off.

Some outbreaks of lead poisoning in domestic animals have been reported in the U.S. in which the apparent source was contamination of pasture or crops by industrial lead operations. Horses are more sensitive and may die after an exposure of 2.4 mg/kg per day for several weeks. It should be noted that horses often ingest soil lead because they pull forage out by the roots and eat both roots and the soil. Cattle rarely do that.

No injuries to man or animals have ever been reported from consuming crops grown near highways. Since unleaded motor fuels will become more available with time, perhaps the problem (or potential problem) will have been averted.

Zinc, Cadmium and Copper

Soil contamination from these metals occurs in urban areas, along highways, near ore smelters and foundries, and from certain pesticides and phosphate fertilizers. Contamination by zinc and copper has been known to occur for decades. However, cadmium contamination, particularly that occurring along highways from automobile tires, has only recently been recognized.

Zinc deposition rates near a smelter in Pennsylvania were found to be 187 to 561 lbs. per acre per year in a recent study. Although cadmium deposition rates were not measured, they were calculated to be about 3 lbs. per acre per year. The study also showed that the soil downwind from the source presently contains 12,000 lbs. per acre of zinc and 160 lbs. per acre of cadmium. About 85 to 95% of the zinc and 95% of the cadmium was found to have been retained in the top 15 centimeters of the soil in high contamination areas.

Foliar concentrations of zinc, cadmium, and copper on oak seedlings grown in contaminated soils were 2,120, 38, and 33 ppm, respectively, while those grown in uncontaminated soils were 155, 0, and 16 ppm, respectively. Trees growing near the source contained up to 4,500 ppm of zinc and 70 ppm of cadmium. Foliar symptoms observed in the same area included interveinal chlorosis by the middle of the growing season, followed by reddening and yellowing.

Mercury Vapors

The phytotoxicity of mercury vapor has been known for nearly 200 years. Reported incidences of injury to plants are very infrequent in spite of the existence of many sources. Unlike other heavy metals, mercury is liquid at normal ambient temperatures, and it is its vapors that are toxic. However, in order for injurious concentrations to occur, the metal must be present in the same confines as the sensitive vegetation. Thus, many sources that emit directly to the atmosphere are not important from a vegetation damage standpoint because the vapors are quickly dissipated. Examples of such sources include primary and secondary ore processing; manufacture of paints, agricultural chemicals, and pharmaceuticals; laboratory and instrument sources; and the combustion of coal, oil, and refuse.

Plant damage has usually occurred as a result of a few very specific emissions. For example, flowers in a commercial greenhouse were injured when mercuric chloride was added to the soil to kill earthworms. In another greenhouse incident, roses were injured because the interior of the greenhouse was painted with a mercury-base paint.

Levels of mercury necessary to cause injury have rarely been determined, and only a general idea of sensitivity is available (Table 6-8). It has been demonstrated that floral parts of plants are more sensitive than leaves, and older leaves are more sensitive than immature ones.

Mercury injury on red roses consists of brown spots on the leaves, and is accompanied by leaf abscission, especially of older, but still active, leaves. The flower petals from partially opened buds turn brown while the young buds turn brown and fall without opening. Fully opened flowers may turn a characteristic faded pink, and stamens may be killed.

Peach seedlings have been found to be very sensitive to mercury vapors. Injury first appears as a fading and subsequently a browning of interveinal tissue on older leaves. The young leaves are the most tolerant. Abscission can occur, sometimes without leaf markings of any kind.

Relative Sensitivity of Plants to Mercury Vapors

Investigators have provided information regarding relative sensitivity of plants to mercury vapors. Table 6-8 is compiled from the sources listed under *Selected References.*

Table 6-8

Relative Sensitivity of Various Plants to Mercury Vapors

Sensitive	*Intermediate*	*Tolerant*
Bean	Azalea	Aloe
Butterflyweed	Basswood	Jersusalem Cherry
Cinquefoil	Begonia	Croton
Boston fern	Camelia	Chinese holly
Holly fern	Columbine	Ivy
Hydrangea	Cosmos	Sarcococca
Mimosa	Cotoneaster	
Oxalis	Forsythia	
Privet	Fuchsia	
Sunflower	Geranium	
Willow	American holly	

Table 6-8 (Continued)
Relative Sensitivity of Various Plants to Mercury Vapors

Sensitive	*Intermediate*	*Tolerant*
	Calla lily	
	Easter lily	
	Japanese maple	
	Oak	
	Peach	
	Persimmon	
	Privet	
	Salvia	
	Saxifrage	
	Strawberry	
	White Pine	
	Tobacco	
	Tomato	
	Viburnum	
	Vinca	

Note: Most of the information on mercury was taken from:

Heck, W. W., Daines, R. H. & Hindawi, I. J. Other phytotoxic pollutants. In: Jacobson, J. S. & Hill, A. C., eds. *Recognition of Air Pollution Injury to Vegetation: A Pictorial Atlas.* Pittsburgh: Air Pollution Control Association, F1-F24, 1970.

Leaf Tissue Analysis

There are several methods available for selectively measuring the presence of heavy metals in plant tissues. One should consult chemical references for the method of choice and approved methods.

SECTION 8. PARTICULATE MATTER

Introduction

A significant fraction of the pollutants found in urban atmospheres consists of small particles. Such particulate matter varies greatly in chemical composition and consists of multi-molecular assemblies that may range in complexity from salt crystals to living cells. From the standpoint of vegetation damage, this class of pollutants is relatively unimportant.

Sources

Combustion products and photochemical aerosols make up a large fraction of the particles in the atmosphere, but these are usually very small in size. Larger particles are contributed by local soil, fine dusts, and sea salt along the coast. Industrial sources of dust include incinerators, cement plants, limestone quarries, industrial furnaces, paper mills, etc.

Atmospheric Chemistry

Dust particles falling on leaves seldom cause injury. Particles in the atmosphere participate in chemical reactions with the surrounding gases and with each other. Particles in a humid atmosphere become surrounded by a water film and water soluble gases like HF or SO_2 can become dissolved in this film. The presence of SO_2 in the water makes the water acidic and, if the acidified particle lands on a leaf, it may burn a hole through the leaf tissues. In humid air, moisture droplets containing dissolved gases reach a critical size and precipitate.

Symptomatology

Dusts may be harmful, harmless, or indirectly beneficial to vegetation depending on their composition. Injury may result from the deposition of a relatively thick crust of lime and the toxic effects of alkaline solutions from cement dust (Plate 6-15). Dust may reduce fruit set by preventing pollen germination on the stigma. Reduction in the number of leaves and stunting of alfalfa has also been reported in heavily contaminated fields. Cement dust may also result in beneficial effects, however, through changes in the soil reaction. With the existing pollution control equipment though, such effects need not occur in the U.S.

Iron oxide dust from sintering plants has caused problems during harvest of vegetable crops because of the abrasive effect of the dust on harvesting machinery and on the surfaces of the plant material.

Limestone dust may accumulate on vegetation as cement dust does (Plate 6-16). However, limestone does not adhere to the foliage as cement does. The soil pH may be changed near sources and cause lime-induced chlorosis on broad-leaved plants.

Air Quality Standards

The primary standard for particulate matter is 75 $\mu g/m^3$, annual geometric mean; the maximum 24-hour concentration not to be exceeded more than once a year is 260 $\mu g/m^3$. The secondary standard is 60 $\mu g/m^3$; the maximum 24-hour concentration not to be exceeded more than once a year is 150 $\mu g/m^3$.

SECTION 9. PESTICIDES

Introduction

Pesticides have revolutionized agriculture in the last half of the 20th century in much the same fashion as mechanization did in the first half. Without the use of some of these chemicals today, the production of food and fiber crops would fall catastrophically short of demand. The public would also have to endure many pests that are not now nuisances. It is difficult even to conceive of agriculture without the use of such chemicals when even now agricultural production, on a worldwide basis, cannot meet demand.

Unfortunately, man, in his haste to increase production, has inadvertently introduced billions of pounds of chemicals into the environment, sometimes with little or no appreciation of the long-term consequences. As evidence of their impact on bird and wildlife populations began to accumulate, the public began to question the wisdom of having maximum amounts of food at the lowest possible price without knowing about the hidden costs.

The use of pesticides will not decrease; indeed, it will probably increase. It is urgent, therefore, that the public be better informed of their value but especially of their potential hazard to man in his habitat. Pesticides by themselves are not pollutants; they become pollutants through misuse.

There are approximately 900 chemical species presently being used as pesticides today, with some 45,000 different formulations. Not all of these chemicals fall within the purview of this book. In general, there are three types of problems associated with unwanted effects of pesticides: (1) trace contamination of environmental systems; (2) food contamination; (3) injury to vegetation. Only the last of the three will be discussed here.

There are also desirable side effects associated with pesticides usage. For example, the systemic fungicide benomyl has been shown to be very effective in protecting certain species of plants from ozone injury. Many pesticides and other agricultural chemicals have been shown to be effective in protecting plants from air pollution. These practices have not been adopted commercially yet mainly because of the cost factor.

Sources

The class of pesticide most frequently involved in plant injury is herbicides. About 50 different herbicides are in use today. Not all have caused problems. Damage to non-target vegetation has been most frequent with the phenoxy herbicides, because of their high volatility and tendency to drift. For example, 2,4-D has been known to drift as far as 15 miles from the site of application. Other factors affecting drift include the type of equipment used and the method of application, the form of the material being applied (spray or dust), and the microclimatology of the area.

Potential sources of injurious herbicides, in addition to their normal use (or misuse), include the manufacture, formulation, and packaging processes. No data are available on emission rates. The greatest source is the application process itself. This latter source is interesting because the application of certain classes of pesticides varies with geographic location. Crop insecticides are used most heavily in the southern U.S., while herbicides are used most heavily in the corn belt.

A number of minor sources exist but are impossible to assess. Such sources include garden and home use, burning of pesticide containers, leakage from storage or discarded containers, and transport from other areas via windblown soil particles.

Symptomatology

Since problems have been associated mostly with the phenoxy herbicides such as 2,4-D; 2,4,5-T; and 2,4,5-TP, the discussion of symptoms will be limited to these chemicals. When non-target crop plants are affected by drift, the most common symptom is deformation of leaves, fruits or other marketable portions of the plant. The leaves develop a "lacy" appearance, and the stem or petiole may become abnormally flattened (Plate 6-17). Discolorations may also occur. Cotton leaves, for example, take on a yellow-green, mottled appearance or stippling and vein-clearing may be pronounced. Epinasty is another frequently observed symptom.

One other problem is often encountered with the use of herbicides. Farmers rotate crops in their field in accordance with accepted practices. However, sometimes a crop will become injured or contaminated with residues from previous crop treatments (Plate 6-18). This is most frequently encountered where S-triazine or dieldrin has been used.

Relative Sensitivity of Plants to Pesticides

No relationship has been established between plant injury and concentrations required to cause such injury in the ambient air. Laboratory investigation, however, has provided information on the relative sensitivity of plants to 2,4-D. These are shown in Table 6-9. Since symptom expression is usually of a growth response type, it is difficult to establish threshold concentrations because of the many factors that affect growth. In addition, some of these chemicals promote growth at very low concentrations.

Leaf Tissue Analysis

There are methods available for detecting pesticide contamination of foodstuffs. The methods have to be developed by the time the pesticides are registered. The methods may be quite complex, however, and expensive instruments and training may be required to perform the analysis.

Table 6-9

Relative Sensitivity of Various Plants to 2,4-D

Sensitive	*Intermediate*	*Tolerant*
Apple	Aster	Ash
Birch	Cedar	Bush bean
Box elder	Cherry	Cabbage
Dogwood	Choke cherry	Eggplant
Elderberry	Corn	Pear
Forsythia	Gladiolus	Peony
Grape	Hemlock	Rhubarb
Hickory	Mulberry	Sorghum
Lamb's quarters	Pin oak	
Linden	Red oak	
London plane tree	Peach	
Norway maple	Potato	
Black oak	Privet	
Sorrell	Ragweed	
Sumac	Rhododendron	
Tobacco	Rose	
Tomato	Spruce (Colorado blue)	
Tree-of-heaven	Sweetgum	
Wisteria	Yew	
Yellow wood		
Zinnia		

Source:
Heck, W. W., Daines, R. H. & Hindawi, I. J. Other phytotoxic pollutants. In: Jacobson, J. S. & Hill, A. C., eds. *Recognition of Air Pollution Injury to Vegetation: A Pictorial Atlas.* Pittsburgh Air Pollution Control Association, F1-F24, 1970.

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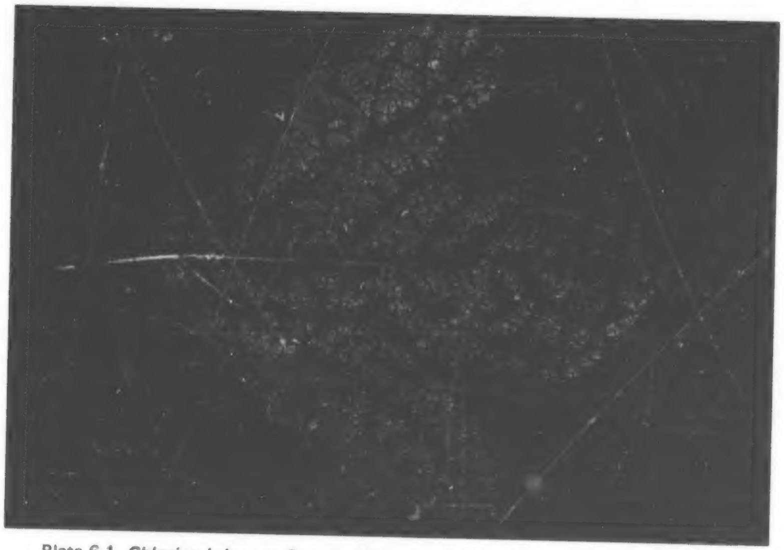

Plate 6-1. *Chlorine Injury to Sassafras Showing Reddish-Brown Interveinal Necrosis*

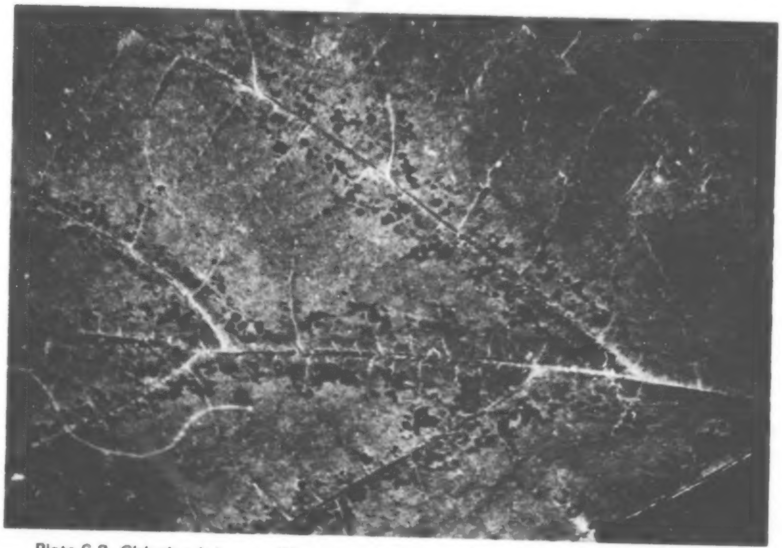

Plate 6-2. *Chlorine Injury to Witch Hazel Showing Necrotic Lesions Along Veins and a Portion of the Leaf Margins*

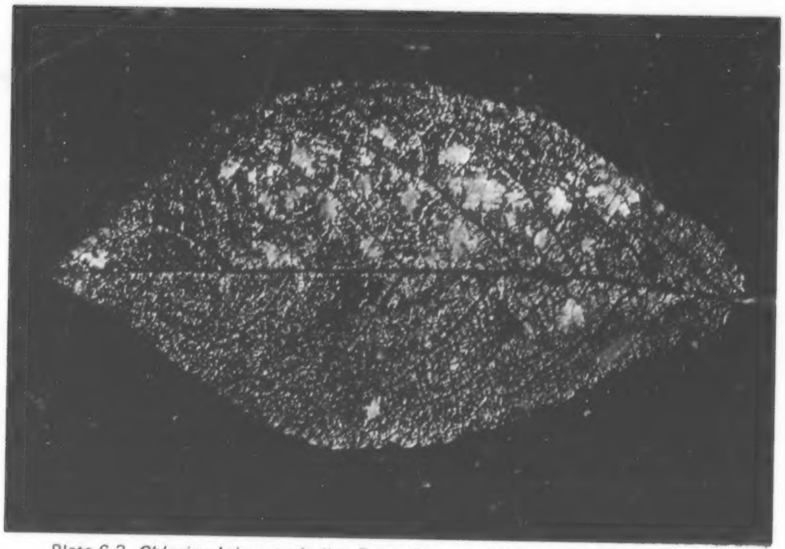

Plate 6-3. *Chlorine Injury to Italian Prune Showing Upper Leaf Surface Flecking*

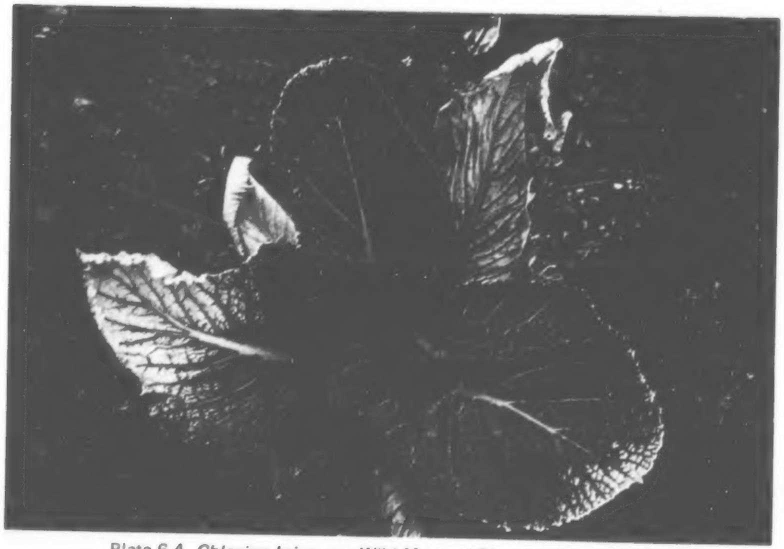

Plate 6-4. *Chlorine Injury on Wild Mustard Showing Bleaching Effect*

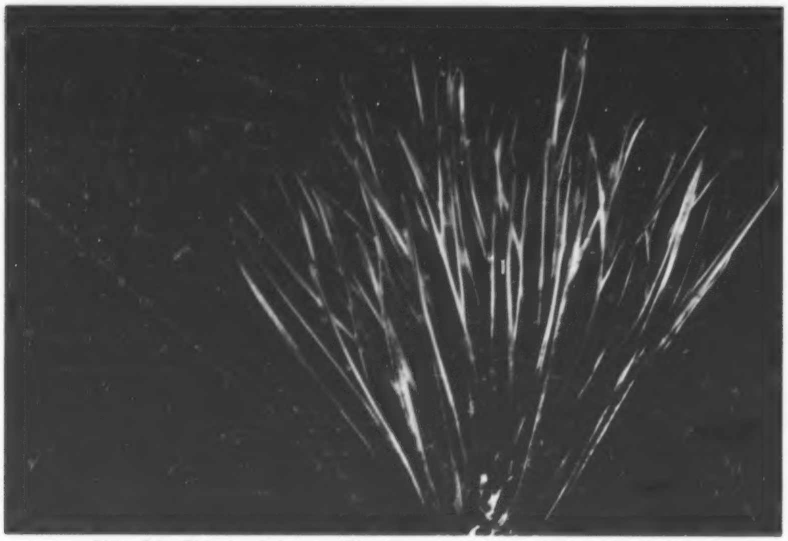

Plate 6-5. *Chlorine Injury on Pine Showing Reddish-Brown Discoloration*

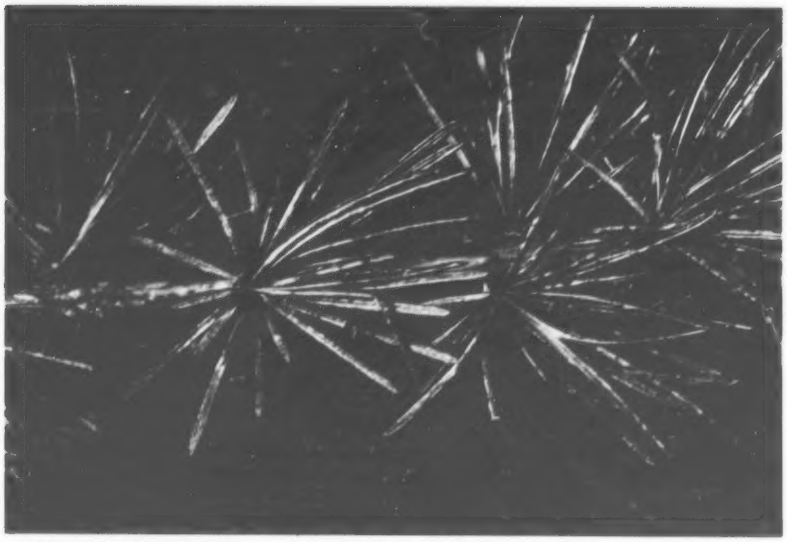

Plate 6-6. *Chlorine Injury on Larch from Chemical Plant Spill. Note Chlorotic Mottle*

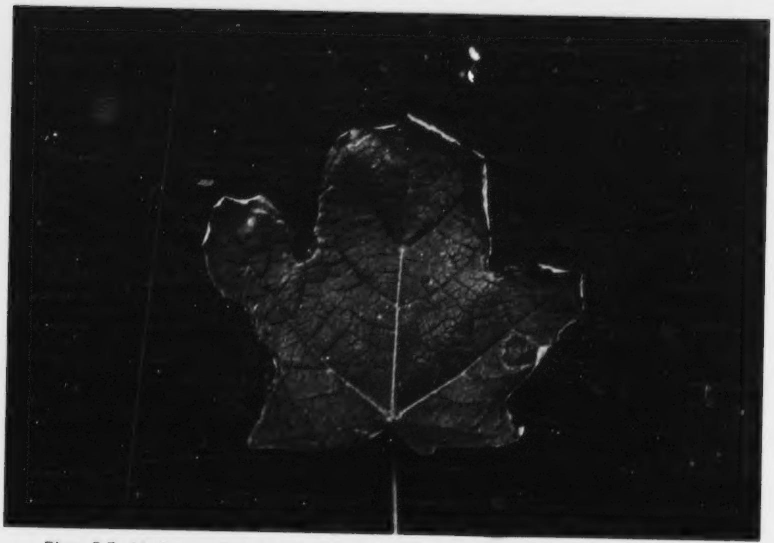

Plate 6-7. *Hydrogen Chloride Injury to Norway Maple Showing Marginal Necrosis*

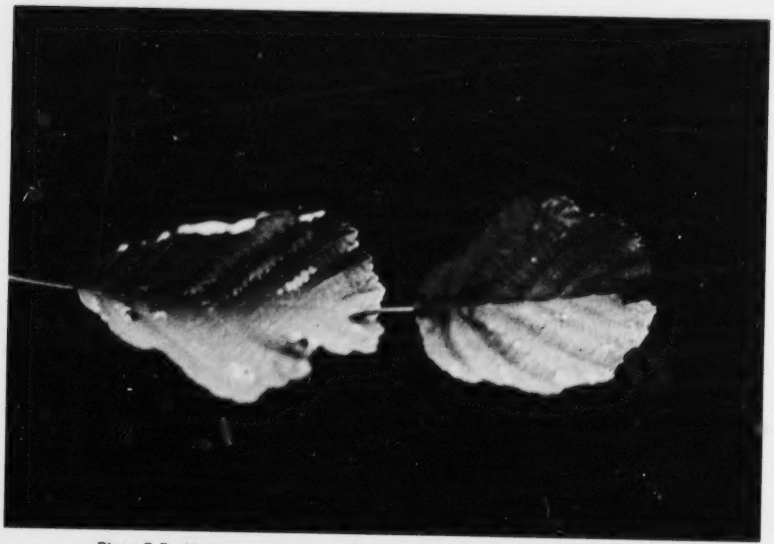

Plate 6-8. *Hydrogen Chloride Injury to European Black Alder Showing Interveinal Necrosis*

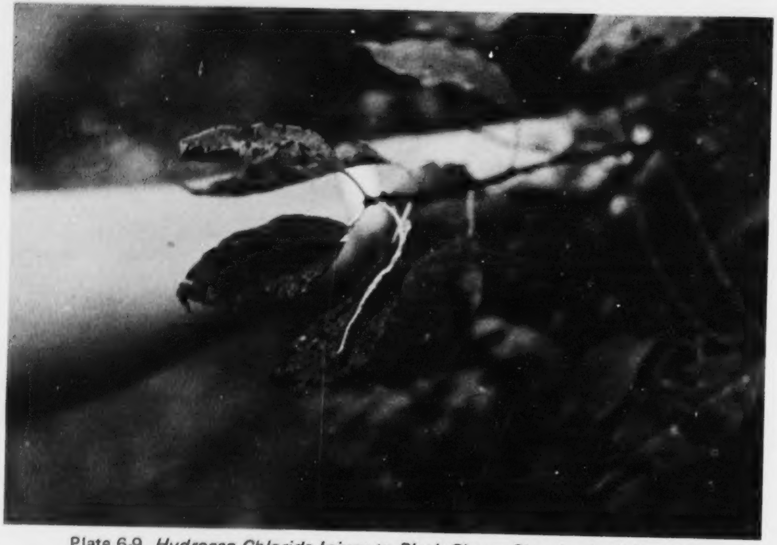

Plate 6-9. *Hydrogen Chloride Injury to Black Cherry Showing Leaf Spotting*

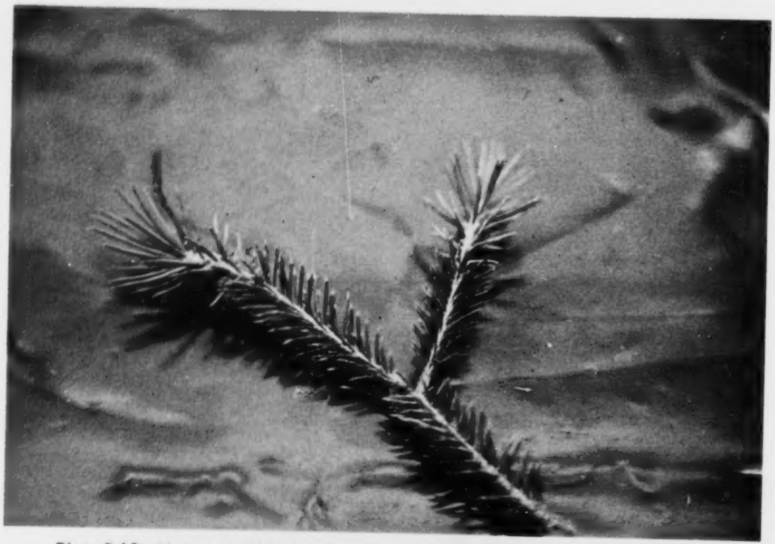

Plate 6-10. *Hydrogen Chloride Injury on Norway Spruce Showing Tip Necrosis*

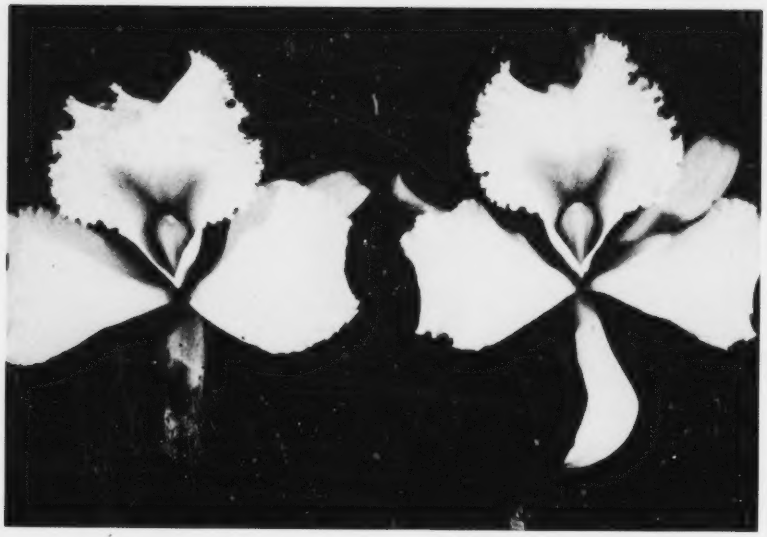

Plate 6-11. *Ethylene Injury on Orchid Showing "Dry Sepal" on Flower at the Left*

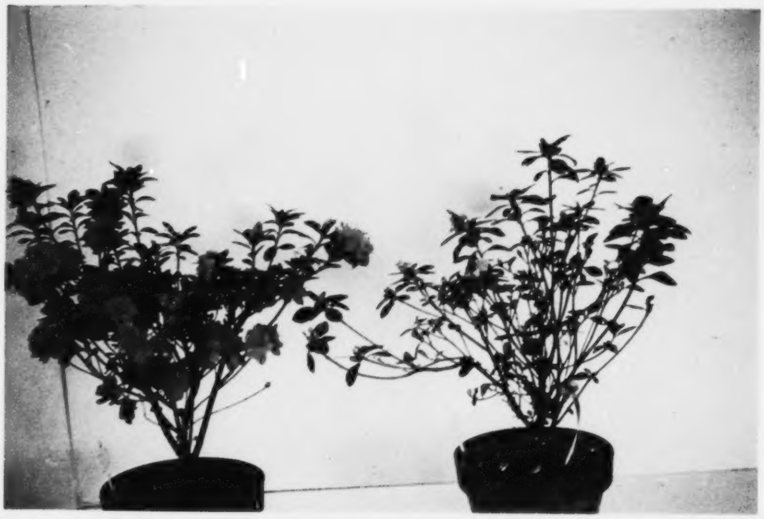

Plate 6-12. *Azalea Fumigated with 2 ppm Ethylene for Seven Days. Note Heavy Leaf Drop and Loss of Flowers. Control Plant on the Left*

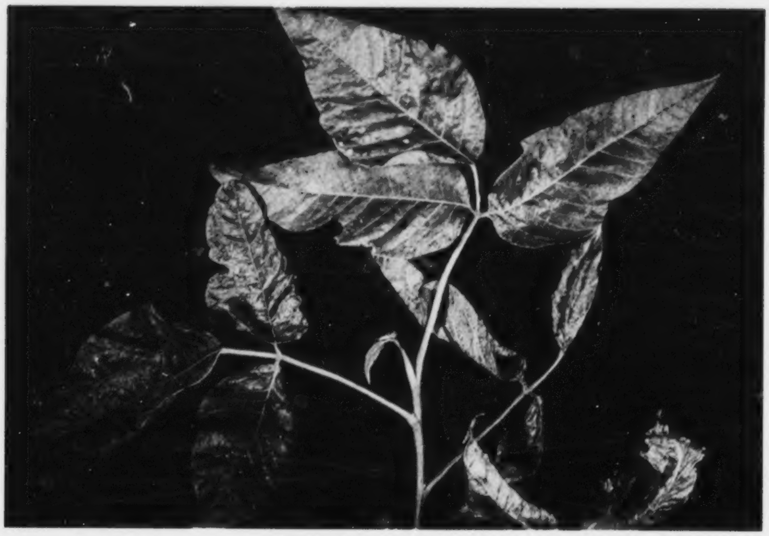

Plate 6-13. *Ammonia Injury on Poison Ivy Showing Blackened Appearance*

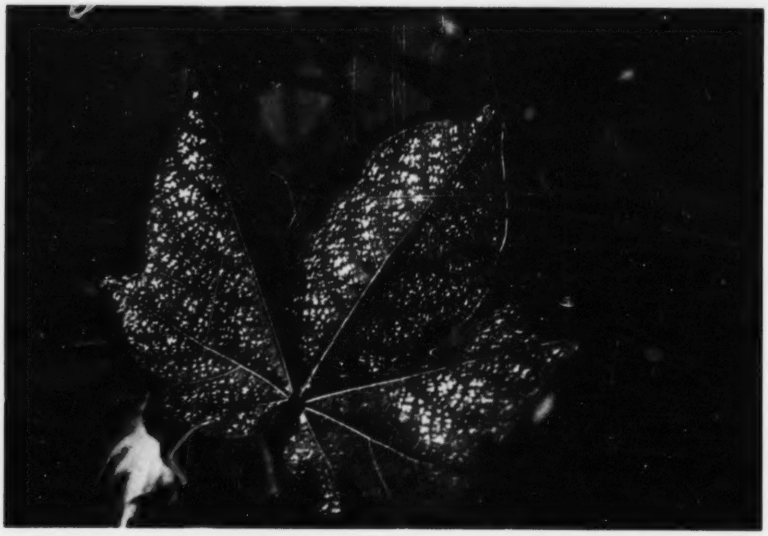

Plate 6-14. *Ammonia Injury on Cotton*

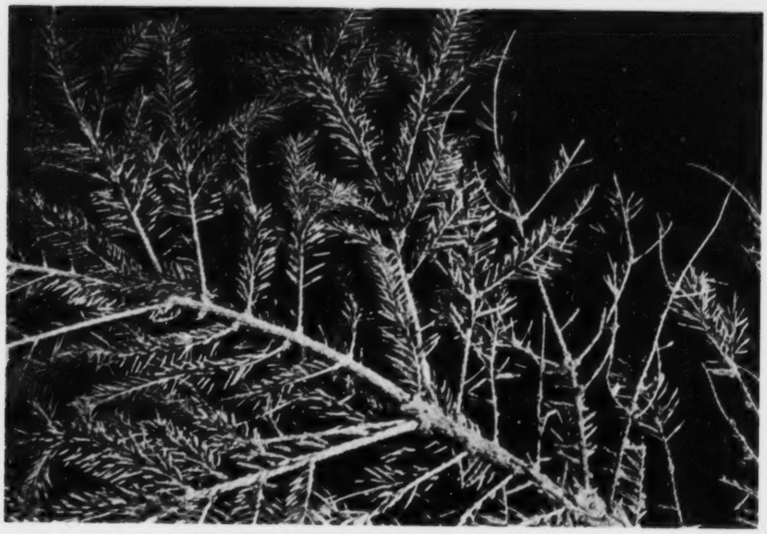

Plate 6-15. *Cement Dust Deposit on Fir Branch*

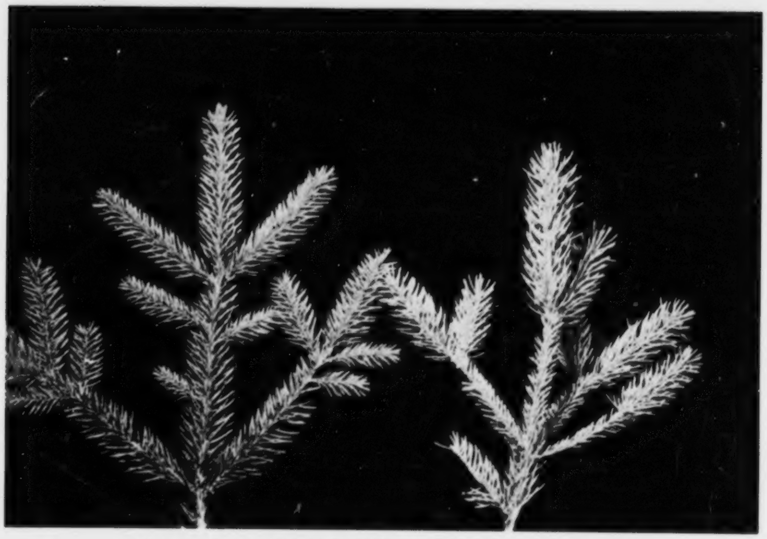

Plate 6-16. *Lime Dust Deposit on Spruce Twigs on the Right*

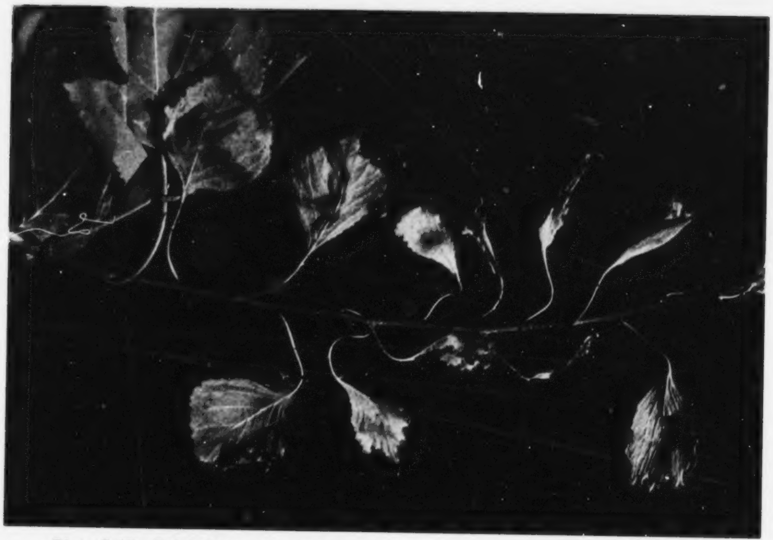

Plate 6-17. *2,4-D Injury to Oak Leaves Showing "Lacy" Appearance of Leaves*

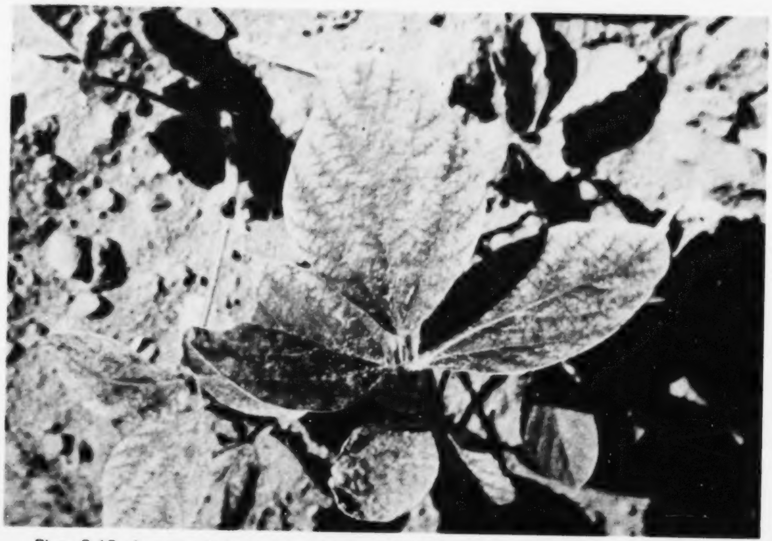

Plate 6-18. *Atrazine Injury on Soybeans. Grain Sorghum was Sprayed with Atrazine During Previous Growing Season*

Chapter 7

INTERACTIONS BETWEEN POLLUTANTS AND BETWEEN POLLUTANTS AND PATHOGENS

More than one air pollutant may be present in the ambient air at the same time. Thus, plants are often exposed to a mixture of pollutants. The effect of these mixtures on plants may be less than, equal to, or greater than the effects of any single pollutant in the mixture. Plant pathogens and air pollutants may also interact to modify the disease response of the plant. The interaction between pollutants will be discussed first.

Pollutant-Pollutant Interactions

Mixtures of gaseous pollutants affect growing plants by: causing injury symptoms on the plants; altering the rate or the type of growth; or altering the productivity of a particular plant species or cultivar. Visible symptoms characteristic of particular pollutants may be caused by different pollutant mixtures. Thus, symptoms produced on various species or cultivars may be confusing.

The first report that one pollutant might affect the response of plants to another pollutant was noted in the early 1950's in California. When researchers equipped greenhouses with water-spray scrubbers to remove sulfur dioxide from the ambient air, they observed increased ambient oxidant injury to Pinto bean. The researchers postulated that either the sulfur dioxide mitigated the effects of the oxidants or the plants were more sensitive at the higher humidity. A few years later, it was found that with an SO_2 to O_3 ratio of 6:1, symptoms of both pollutants appeared. When the ratio was lowered to 5:1, ozone symptoms predominated and no interference from SO_2 was noted. When the ratio was lowered to 4:1, the ozone appeared to interfere with the expected SO_2 injury symptoms. No evidence of increased injury was reported with the pollutant mixtures. No interacting effects were found on gladiolus when fumigated with combinations of SO_2 and HF or SO_2 and hydrocarbons. Likewise, propylene and acetylene did not interfere with the development of foliar symptoms from products of irradiated propylene-NO_2 mixtures. It was found in 1966 that certain cultivars of tobacco were slightly injured when fumigated with 0.24 ppm of SO_2 or 0.03 ppm of O_3 for 2 or 4 hours. However, when the tobacco cultivars were exposed to a mixture of the two pollutants, the level of injury increased to moderate. These results suggested some type of potentiation mechanism or synergism between the pollutants and stimulated new research efforts.

The field observer should recognize that several pollutants may be present either simultaneously, sequentially, or intermittently. Thus in an urban area, with a number of SO_2 sources, plants may be exposed to SO_2 during the early morning hours and to oxidants from photochemical reactions (and possibly SO_2) later during the day. Other pollutants may affect the response if other industries are present in or around the urban area. Only a few of the more obvious pollutant combinations have been studied.

Sulfur Dioxide and Ozone

This pollutant combination has received the greatest attention because of the increase in general SO_2 emissions from mine-mouth power plants and the ubiquitous nature of oxidants (ozone). Some believe these pollutants are causing widespread effects at this time.

In general, foliar symptoms caused by O_3 are easily distinguished from those caused by SO_2. Mixtures of these two gases at concentrations below the injury threshold for SO_2 and in some cases below the threshold for O_3 produce symptoms resembling those caused by O_3 (Plate 7-1). Usually SO_2 symptoms do not appear unless the concentration is well above the injury threshold for SO_2 (Plates 7-2, 7-3 and 7-4).

Table 7-1 contains selected examples of foliar effects of SO_2-O_3 fumigations. In some plant species the effect is *additive* (equal to the additive effect of the single pollutants) while in others the effect is greater or less than additive. No general trend was found to indicate how ratios of the two pollutants would affect foliar response. Symptoms in these fumigations frequently appeared on both leaf surfaces. The undersurface injury was often a silvering and a collapse of epidermal tissue (Plate 7-5) while the upper surface was usually an interveinal necrotic fleck, stipple, and/or pigment accumulation (Plate 7-6).

Table 7-1

Selected Examples of Effects of Sulfur Dioxide and Ozone Mixtures on Foliar Injury

Plant Species	*SO_2/O_3 Conc., ppm*	*Exposure Time, hrs.*	*Mixture Response**
Broadleaf			
Alfalfa	0.1-0.5/0.1	4	+
Bean	1.7/0.19	0.5	+
Onion	1.0/0.1	4	0
Soybean	1.0/0.1	4	0
Spinach	1.0/0.1	4	0
Tobacco	0.45/0.03	2-4	+
Tomato	0.1-0.5/0.1	4	-,0
Narrowleaf			
Hungarian brome grass	1.0/0.1	4	0
Conifer			
White pine	0.1/0.1	8 hr./day 5 day/wk. 8 wks.	+

*Mixture responses are: + greater-than-additive
0 additive
– less-than-additive

Source:
Reinert, R. A., Heagle, A. S. & Heck, W. W. Plant response to pollutant combinations. In: Mudd, J. B., ed., *Effects of Air Pollution on Plants: Chapter II.* New York: Academic Press, 1975.

Sulfur Dioxide and Nitrogen Dioxide

This pollutant mixture is of concern because the two gases are emitted simultaneously in the combustion of fossil fuels and the reduction of ores. Exhaust emissions from automobiles add considerably to NO_2 levels in large urban areas.

The symptoms resulting from this pollutant mixture are similar to those caused by O_3, especially when concentrations of the combined pollutants are near or below the injury threshold of the individual pollutants. Plate 7-7 shows the effect of .10 ppm of SO_2 and .10 ppm of NO_2 on Bel W-3 tobacco on the upper surface of the leaf. A reddish pigmentation or a silvering, not necessarily associated with uppersurface injury, may develop on the lower surface of the leaf as shown in Plate 7-8. Since the symptoms associated with this pollutant mixture are so similar to those caused by ozone, and since ozone injury is so prevalent, identification of the causal pollutant is difficult. It is possible that foliar injury in the field previously reported to be caused by O_3 may have been caused by this pollutant mixture.

Table 7-2 contains selected examples of responses of plants to SO_2/NO_2 mixtures.

Table 7-2

Selected Examples of Effects of Sulfur Dioxide and Nitrogen Dioxide Mixtures on Foliar Injury

Plant Species	*SO_2/NO_2 Conc., ppm*	*Exposure Time, hrs.*	*Mixture Response**
Broadleaf			
Aspen	0.5-0.7/0.15-0.21	2	0
Bean	1.5/1.5	1.17	+
Radish	0.05-0.25/0.05-0.25	4	+
Soybean	0.05-0.25/0.05-0.25	4	+
Tobacco	0.1/0.1	4	+
Tomato	0.05-0.25/0.05-0.25	4	+
Narrowleaf			
Oats	2.4/1.3	1	-

*Mixture responses are:
+ greater-than-additive
0 additive
– less-than-additive

Source:
Reinert, R. A., Heagle, A. S. & Heck, W. W. Plant response to pollutant combinations. In: Mudd, J. B., ed., *Effects of Air Pollution on Plants: Chapter II.* New York: Academic Press, 1975.

In studies of plants grown under semi-arid conditions such as in the southwestern United States, the response of plants to mixtures of SO_2 and NO_2 has been typical of SO_2 injury. The injury, most commonly leaf necrosis, appears within a few days following exposure in the field. On *broad-leaved* species, injury develops as interveinal patches of bifacial necrotic tissue. The color varies with plant species and may appear as tan, gray, brown, yellowish-brown, or rusty brown. Symptoms on *narrow-leaved* species develop in patches, streaks, or spots of white, yellow, or light brown necrotic tissue. On *conifer* species such as pine and juniper, the injury symptoms develop as brown or reddish-brown necrosis of the needle tips and necrotic spots along the needles and scales. Middle-aged and older leaves are consistently more sensitive to injury than the younger leaves. These data suggest that there is no injury potentiation (i.e., no synergism) with this gas mixture under these growing conditions. Much of the injury is due to SO_2.

Sulfur Dioxide and Hydrogen Fluoride

Sulfur dioxide and fluorides may occur together in areas where smelters or aluminum reduction plants are located near coal-fired power plants or other SO_2 sources.

The effects of both of these gases occurring singly are well known. However, fumigations with a mixture of these two gases have resulted in injury to some plant species but not others. Cultivar differences have been reported. For example, continuous fumigation with a mixture of 0.8 ppm SO_2 and 2.3 to 17.1 ppb of HF for 23 days resulted in additive decrease in linear growth of branches and reduction in leaf area but no visible injury or leaf abscission on Koethen sweet orange. When Satsuma orange was exposed to the same mix for 15 days, however, the fumigation resulted in foliar injury that was less than additive and no growth effects were observed.

Selected examples of plants' response to this gas mixture are presented below in Table 7-3.

Table 7-3
Selected Examples of Effects of Sulfur Dioxide and Hydrogen Fluoride Mixtures on Foliar Injury

Plant Species	*SO_2/HF Conc., ppm/ppb*	*Exposure Time*	*Mixture Response**
Broadleaf			
Bean	0.08/0.6	27 days	not defined
Koethen sweet orange	0.8/2.5-13	23 days (10-day interruption)	0
Satsuma orange	0.8/2.5-13	15 days (10-day interruption)	–
Tomato	0.5/5.0	4 hrs., 2 day/wk.	not defined
Narrowleaf			
Barley	0.08/0.8	27 days	+
Corn	0.08/0.6	27 days	+

*Mixture responses are: + greater-than-additive
0 additive
– less-than-additive

Source:

Reinert, R. A., Heagle, A. S. & Heck, W. W. Plant response to pollutant combinations. In: Mudd, J. B., ed., *Effects of Air Pollution on Plants: Chapter II.* New York: Academic Press, 1975.

Pollutant-pollutant interactions are quite complex and our knowledge of these interactions at the present time is, at best, fragmentary. However, their importance may prove to be far greater than the effects of single pollutants. Field detection of these interactions may be very difficult. However, many states now have comprehensive monitoring programs which could provide the type of information needed to establish pollutant levels at the air basin level.

Pollutant-Pathogen Interactions

Air pollutants may interact with plant pathogens in two ways: (1) they may affect the pathogen during some stage of its life cycle; (2) they may alter the susceptibility of plants to a given pathogen. The effects of these interactions may be harmful or beneficial to the plant. Relatively little is known about these interactions. However, the available literature on this subject has been summarized recently in a review article by A. S. Heagle (see *Selected References*).

In general, a decrease in the incidence of disease in the presence of SO_2 in ambient air has been reported. Sulfur dioxide, for example, can effectively reduce the incidence of black spot of roses, and can also reduce the size of the fungus lesions if it does develop. However, it has also been reported that needle cast disease on scotch pine can be more severe at some distance from an SO_2 source because the fungus can utilize the sulfur in the air.

Ozone has been shown to decrease wheat rust and powdery mildew infections and increase the incidence of Botrytis leaf spot on potato and geranium leaves. Fluoride as HF can inhibit bean rust, bean halo blight, and tomato early blight infections. Preliminary work with cement and limestone dusts indicates that these two pollutants can enhance bacterial colonization of leaves.

There is very little information that would be helpful in field diagnosis. Selected examples of pollutant-pathogen examples are presented in Table 7-4.

Table 7-4
Selected Examples of Pollutant-Pathogen Interaction

Disease affected, by pollutant	*Pollutant Dose*	*Changes in Disease Incidence*
SO_2		
Stem rust of wheat	ambient	decrease
Needle casts on larch, pine, juniper	ambient	decrease
Tan leaf spot (maple)	ambient	decrease
Apple scab	ambient	decrease
Powdery mildew (oak, lilac)	ambient	decrease
Black spot of rose	ambient	decrease
Dwarf mistletoe (conifers)	ambient	decrease
Armillaria root rot	ambient	increase
Needle cast on spruce	ambient	increase

Table 7-4 (Continued)

Selected Examples of Pollutant-Pathogen Interaction

Disease affected, by pollutant	*Pollutant Dose*	*Changes in Disease Incidence*
O_3		
Wheat stem rust	0.06 ppm, 6 hrs.	decrease
Powdery mildew (barley)	0.25 ppm, 8-12 hrs. after innoculation	decrease
Botrytis leaf spot	ambient	increase
Tobacco mosaic virus in tobacco	Tobacco exposed to 0.30 ppm, 6 hrs.	increase on pinto beans
HF		
Bean rust	300 ppm in leaf tissue	decrease
Bean halo blight	300 ppm in leaf tissue	decrease
Tomato early blight	300 ppm in leaf tissue	decrease
Tobacco mosaic virus	100-300 ppm in leaf tissue	increase on pinto beans

Source:
Heagle, A. S. Interactions between air pollutants and plant parasites. *Annual Review of Phytopathology,* 11:365-388, 1973.

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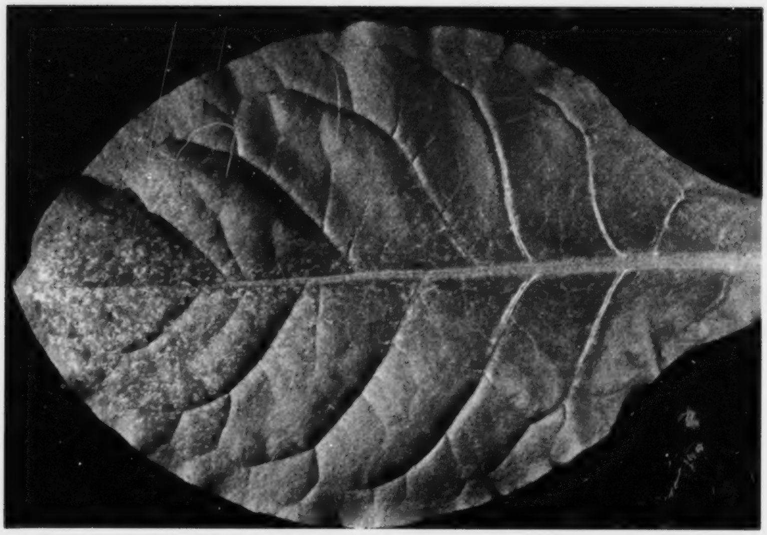

Plate 7-1. *Bel W-3 Tobacco Exhibiting Injury from Fumigation with .05 ppm O_3 and .10 ppm of SO_2. Note the Similarity to Ozone Injury*

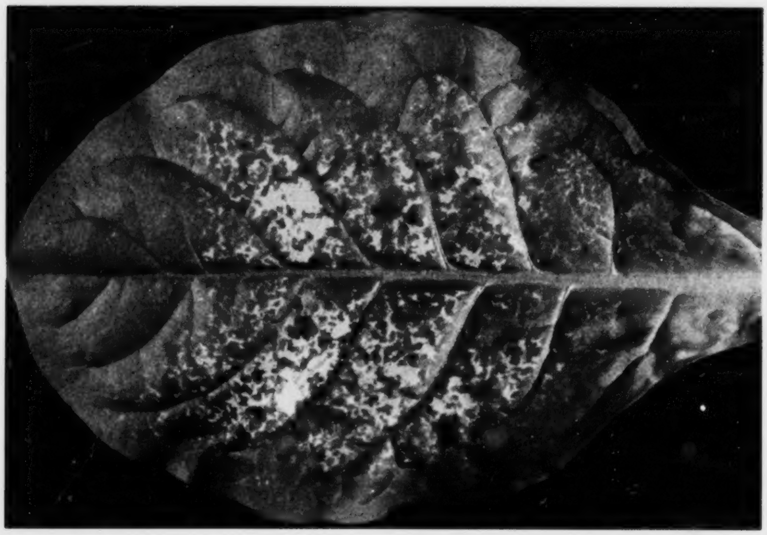

Plate 7-2. *Injury to Bel W-3 Tobacco Fumigated with .05 ppm of O_3 and .50 ppm of SO_2. In This Case the Injury More Closely Resembles SO_2 Injury*

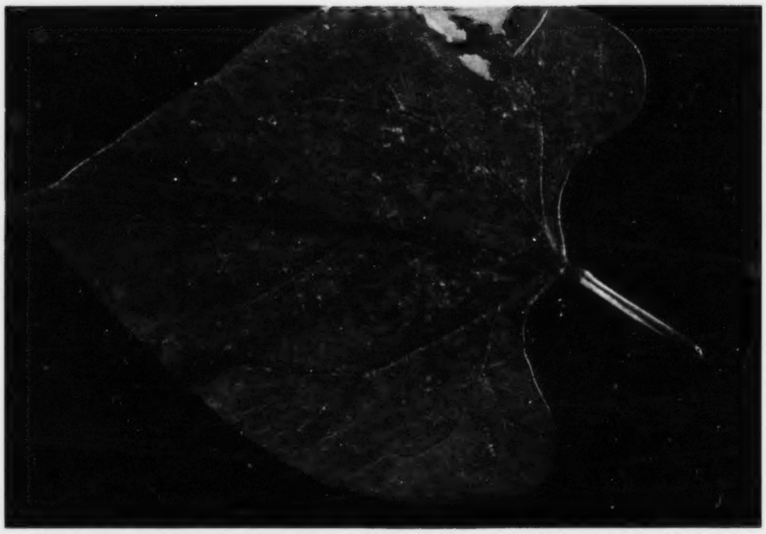

Plate 7-3. *Pinto Bean Fumigated with .15 ppm of O_3 and .50 ppm of SO_2. Note the Almost Complete Absence of Bleaching and the Slight Purplish Discoloration*

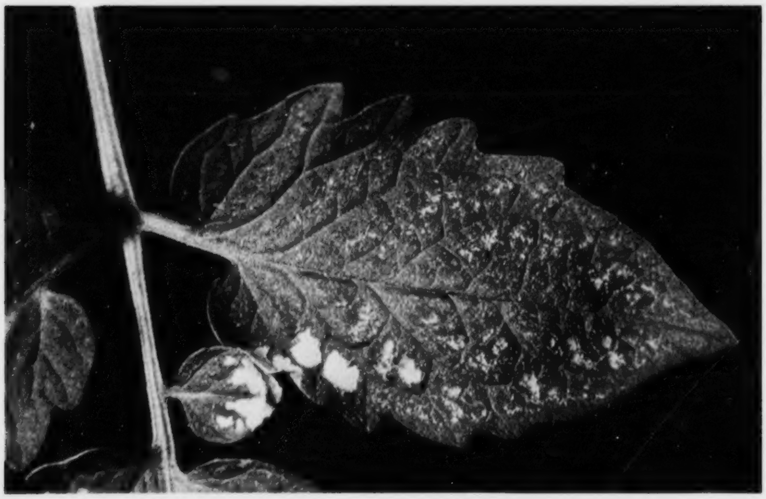

Plate 7-4. *Bleaching of Tomato Leaf as a Result of Fumigation with .05 ppm of O_3 and .25 ppm of SO_2*

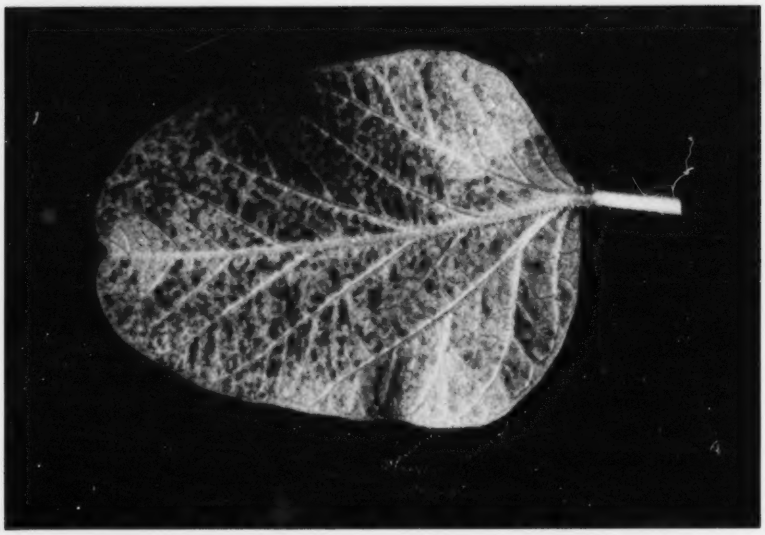

Plate 7-5. *Lower Surface of Soybean Leaf Fumigated with .10 ppm of O_3 and .50 ppm of SO_2. Note the Difference in Symptoms from Plate 7-6*

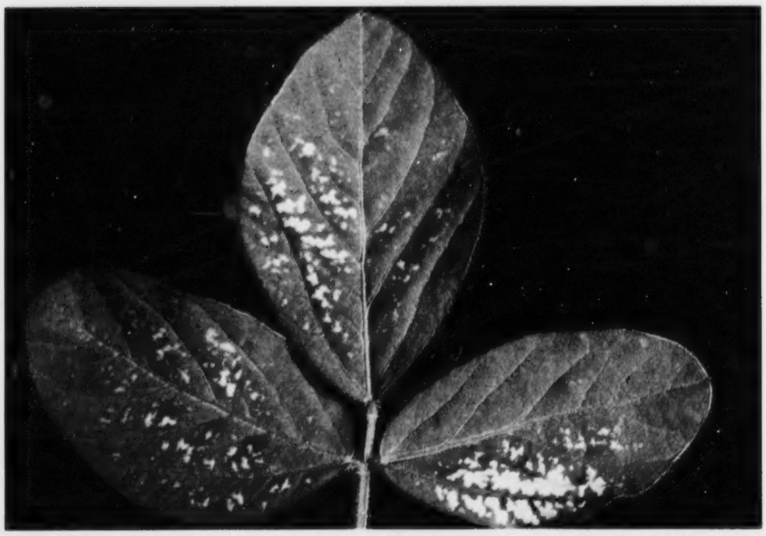

Plate 7-6. *Soybean Leaf Fumigated with .10 ppm of O_3 and .50 ppm of SO_2*

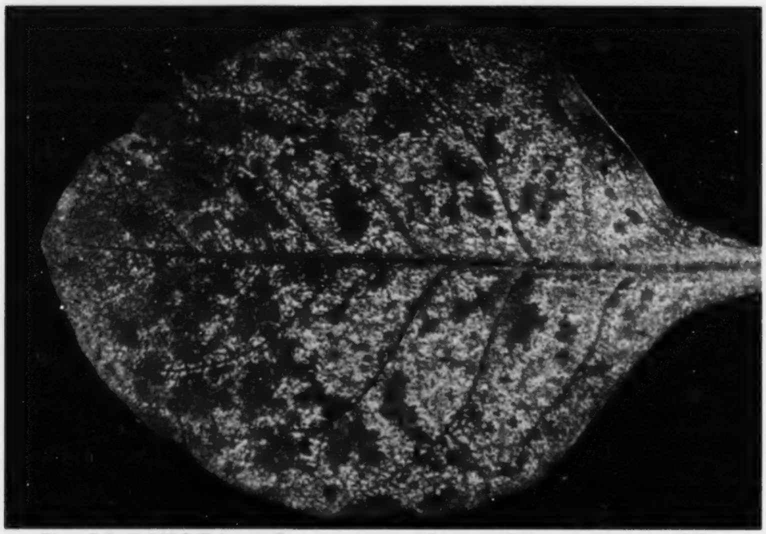

Plate 7-7. *Bel W-3 Tobacco Fumigated with .10 ppm of SO_2 and .10 ppm of NO_2*

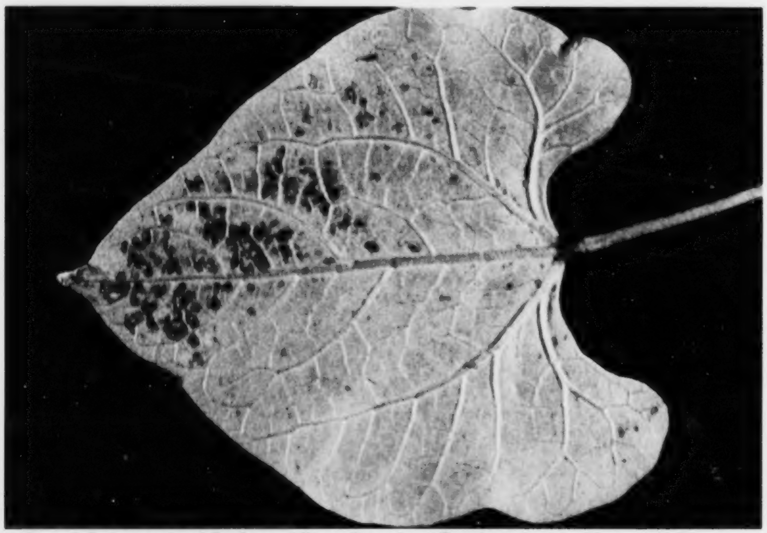

Plate 7-8. *Lower Leaf Surface of Pinto Bean Leaf Fumigated with .05 ppm of SO_2 and .10 ppm of NO_2*

Chapter 8

MIMICKING SYMPTOMS

The preceding chapters may suggest that diagnosis of air pollution injury is a relatively simple matter, particularly if the sources of pollution are identified in a given area. In some situations diagnosis may be simple and straightforward, in others it may be very perplexing. Plants are affected by biotic and abiotic agents (Table 8-1). One must be aware that some of the foliar symptoms associated with these agents may resemble or "mimic" air pollution symptoms. Plant diseases are common and, even though foliar injury may not resemble air pollution injury, it may be associated with air pollution by the public. The type of injury depicted in Plates 8-1, 8-2, 8-3 and 8-4, for example, do not mimic pollution symptoms. However, an uninformed public may attribute this injury to exhaust fumes because of the proximity of the trees to automotive travel.

Table 8-1

List of Agents That Can Induce Plant Diseases*

I. Abiotic Agents
- Lack of or Excess of:
  - water
  - temperature
  - nutrients
  - soil acidity
  - de-icing salts
  - pesticides
  - construction injury and cultural practices
  - air pollutants

II. Biotic Agents
- Fungi
- Viruses and mycoplasmas
- Bacteria
- Nematodes
- Insects

III. Teratogenic Agents
- Genetic disorders

*Response to some of these agents may mimic symptoms of air pollution injury.

To distinguish between air pollution symptoms and mimicking syptoms, one must have knowledge of plant diseases and their causes.

A disease is an interaction between a plant, a pathogen, and the environment which results in an altered or detrimental condition in that plant. Diseases may be induced by fungi, nematodes, bacteria, viruses, insects, mineral imbalances, or various physical stresses. The purpose of this chapter is to create an awareness for the many potential causes of disease. Plant disorders are usually grouped into three categories: those caused by abiotic agents, such as air pollutants, mineral deficiencies, etc.; those caused by biotic agents, such as fungi, bacteria, etc.; and those caused by teratogens.

Abiotic Agents

Water

The symptoms of prolonged drought are easily recognized and should be easy to diagnose. Complete and permanent collapse of succulent tissues and dieback, or even death, of entire plants may occur. Typical wilt symptoms may be in evidence. Water will correct the situation if the permanent wilting point has not been reached.

The symptoms associated with short periods of drought or sudden, severe droughts followed by adequate moisture are less easily recognized. Marginal scorch of foliage will occur where adequate moisture levels have not been maintained. An orange coloration will occur on affected tissues of maples. Three separate types of drought-induced necrosis are: (1) leaf tip necrosis with a sharp demarcation line between affected and healthy tissues; (2) leaf tipburn with a chlorotic area separating the necrotic from the healthy tissues; and (3) dull brown or bronzed interveinal lesions that are irregular in outline.

These same types of symptoms may occur after fill has been placed around trees. The resulting changes in water flow patterns can cause local "droughts," especially for hydrophyllic species such as willow, rhododendron, and sycamore (Plate 8-5). Purple coloration of dogwood in July due to drought, and insect boring of the stem could be confused with O_3 or SO_2-induced color changes. The normal cast of two- and three-year-old needles from conifers has often been attributed erroneously to air pollution injury. Symptoms expressed by nearby pollutant-sensitive species should assist in eliminating natural senescence as a factor for consideration. Also, natural senescence occurs only toward the end of the growing season.

Excessive amounts of water will also lead to plant injury. Symptoms are usually associated with man-made changes in grade levels around construction sites or adjacent to new highways where streams have been blocked without the use of drainage culverts. Symptoms include dwarfing of leaves, general chlorosis of all foliage, dieback and death. Affected plants may die gradually and the cause can be readily identified by an examination of drainage conditions in the area.

Temperature

Frost, either in the late spring or early fall, will injure sensitive plant tissues. Late spring frosts are the most damaging since the young foliage is succulent at that time of year. Diagnosis is easy because the frozen tissues take on a blackish-brown coloration. Mild freezes or even cool temperatures may cause slight tissue collapse and/or chlorosis that may resemble air pollution induced symptoms such as severe SO_2 injury (Plate 8-6). Weather records, and personal observations can assist in identifying injury caused by late spring frosts. Premature autumn coloration and leaf drop in several species of plants, initiated by early fall frosts, are common. Green foliage may actually drop from the tree, and that which remains may have light-brown or other colored margins.

Many ornamentals, shade trees, and forest tree species are susceptible to winter injury. The soil water becomes frozen due to extended periods of sub-freezing temperatures, and if the water remains frozen during subsequent warm periods, the foliage may lose water and desiccate. The affected leaves may remain green throughout the winter months or turn yellow and brown the following spring (Plate 8-2). The entire leaf may be killed. On thick-leaved species, such as magnolia, only the leaf tips may be affected. Deciduous plants usually suffer less from winter injury, but orchard trees and even entire forest stands have been killed.

Injury may occur to many plant species even though adequate moisture is available in the soil during periods of extremely high temperature combined with windy conditions. This type of injury rarely occurs in open fields, but it is common in urban areas where higher than normal temperatures occur. The injury symptoms are characterized by a marginal scorch of leaf tissues, with a fairly sharp line developing between affected and healthy areas. The symptoms appear on the periphery of the crown of trees. The symptoms may resemble those induced by fluorides.

Heat-induced defoliation may occur near such activities as street paving with heat-releasing processes and refuse burning (Plate 8-1). Brief exposures to high temperatures may result in a temporary mild chlorosis in young foliage. These symptoms may disappear as the season progresses. Periods of cool, wet weather during the spring that are followed by sudden extremely high temperatures commonly lead to increased amounts of heat injury. Heat injury may also occur on plants that have been growing in shaded areas that are either transplanted into or suddenly exposed to direct sunlight.

Nutrients

There is a plethora of information on the symptoms associated with the lack or excess of nutrient supply to plants. The effect of either of these two extremes is sometimes difficult to diagnose and may require the use of soil and/or plant tissue chemical tests (Plates 8-7 and 8-8).

Symptoms of a specific nutrient deficiency may occur on all parts of a plant or on specific parts, such as on flowers, fruits, or most commonly on the foliage. The coloration involved and the plant part affected are often the best means of determining the element involved. Major lists of nutrient disorders are available. Some of these symptoms are similar to air pollutant symptoms.

Soil Acidity

Unfavorable soil pH may result in mineral imbalances in the soil solution and these imbalances are usually manifested in the foliage as nutrient excesses or deficiencies. For example, the presence of either low or high pH may lead to either the release of minerals such as aluminum that become toxic to the plant, or to the tying up of others, such as iron or phosphorus, that are essential for plant growth. Soil analyses should be conducted in field-grown crops where injury is present and subsequent comparisons should be made with known optimum levels for the specific crop involved.

In the eastern U.S., soil acidity is the most common cause of yellowing of foliage in some crops, resulting in stunting of overall plant growth, loss of leaves, and sometimes death of entire plants or crops. Excessively high pH levels, as found in the alkaline soils of the west, lead to very similar symptom patterns.

One example of suspected air pollution injury that was finally and correctly attributed to improper soil pH conditions occurred near a major source of several air pollutants in east-central Virginia during 1970. Nearly half of the seedlings in a large tree nursery of loblolly pines had severe chlorotic mottle. The nursery is located near industrial sources of SO_2, F, and NO as well as being located in the urban center of the state. Symptoms appeared similar to SO_2 and F injury, but soil pH was found to be less than 4.0. Loblolly pine grows best in soils of pH 5.6 to 6.0 and a simple liming practice alleviated the symptoms.

De-icing Salts

Many thousands of miles of highway in sections of the United States where winter is severe are salted during snow storms to melt ice and snow. During the summer months, salts are used to settle dust on unpaved roads. Salts such as calcium chloride ($CaCl_2$) and sodium chloride (NaCl, common table salt) are used in this practice. When these salts accumulate in the soil adjacent to highways, plants growing there are injured. Hundreds of roadside maples in New England alone have been severely injured or killed by salt (Plate 8-3). Salt injury symptoms are somewhat similar to those induced by F or SO_2. These include marginal leaf scorch of broad-leaved foliage and needle tipburn of conifers. The location of the injured plant in relation to the roadside, and knowledge concerning the use of salt along such highways, assist in proper diagnosis of this cause of injury.

Salt spray from de-icing salts will also injure evergreens growing adjacent to roadsides (Plate 8-4). An uninformed public may attribute this injury to auto exhaust simply because of the location of the plants.

Pesticides

Certain pesticides cause symptoms on various plants that also resemble ozone injury. For example, the miticide Kelthane causes injury on soybeans that resembles ozone injury (Plate 8-9). Other ozone-like symptoms may be caused by fertilizer excess, nutrient deficiencies, etc. Some pesticides, when applied in excess or under improper temperature or moisture conditions, may cause burn of foliage that may resemble air pollution injury. Knowledge of previous pesticide applications, amounts, and conditions under which they were applied should assist in diagnosis. Symptoms such as interveinal necrosis, leaf spotting, tipburn, and death are all possible. Lawn fertilizers that contain herbicides are often misused as tree and shrub fertilizers, and consequently the herbicide ingredient causes foliate symptoms of chlorosis, leaf scorch, and defoliation.

Construction Injury and Improper Cultural Practices

The construction of homes in wooded areas injures many trees that are retained as shade trees. Direct stem injury is easily diagnosed but the effects of root injuries and subsequent infection by root-invading fungi may not appear for several years. Symptoms of root injury include dieback of twigs, small leaves, marginal leaf scorch, premature leaf coloration and defoliation.

Improper cultural practices such as poor planting procedures, the planting of species not adapted to specific locations, and poor maintenance of overall plant vigor may alone or in combination with many other factors cause symptoms similar to air pollutant injuries.

Air Pollutants

The symptoms induced by a specific air pollutant may not be unique to that pollutant alone. Several pollutants, including SO_2, F, and O_3, will cause a tipburn on conifers depending upon the dosage and species of conifer involved. SO_2, O_3, Cl_2, and HF all produce similar symptom patterns on many species of plants. In fact, a number of biotic and abiotic agents can produce symptoms on conifers that mimic those produced by HF. Necrosis of the tip of the needle can result from exposure to other air pollutants, such as Cl_2, SO_2 or O_3, although the initial stages of injury may not be the same as those induced by HF. Low temperatures as well as stresses induced by high temperatures and drought also may produce tipburn. Chemicals applied to, or migrating into, the soil, such as de-icing salt, boron herbicides, and excess fertilizers, can induce tipburn. Insects, such as scale insects, can induce a fluoride-like tipburn of pine needles. Indeed, when tipburn occurs it is so non-specific that for the correct diagnosis one must rely on the elimination of other causes, examination of symptoms on other plants, and chemical diagnosis, as well as the likelihood of an effect of fluorides from a known source.

Biotic Agents

Plant diseases may be caused by fungi, bacteria, viruses, mycoplasmas, and nematodes. Some of the symptoms associated with these diseases closely resemble those caused by some pollutants. Insect injuries may also be confused with air pollutant-induced symptoms. Only a brief discussion of the general symptomatology associated with each will be presented below. Examples of each will be used to illustrate the importance of recognizing these causal agents. Although these organisms are capable of causing symptoms on most plant parts, the symptoms expressed on leaves most often mimic those caused by air pollutants.

Fungi

Leaf-infecting fungi may cause early symptoms that mimic air pollutant-caused symptoms. The initial symptoms expressed by needlecast of conifers, oak and sycamore anthracnose, early blight of tomato, and several leaf-spotting diseases of ornamentals are just a few examples (Plate 8-10). However, as the disease development progresses, the cause becomes obvious. Initial symptoms of these diseases include chlorotic spots on the leaf surface which eventually become necrotic. Fruiting bodies of the organism erupt from the necrotic tissues and become visible to the unaided eye.

Roots invaded by fungi such as *Phytophthora spp.* on vegetables, ornamentals, or *Fomes annosus* and *Armillaria mellea* on forest and shade trees will cause above-ground symptoms such as chlorotic and necrotic foliage, dieback, and death. Mild chlorosis may occur from a light infection by root-invading fungi such as *Paicylomyces* on boxwood.

Wilt-inducing fungi such as *Fusarium oxysporium* f. *lycopersici* that cause wilt of tomato may also induce symptoms of air pollutant injury. However, much of the fungus-caused diseases are easily distinguished from air pollution injury by culturing and identifying the fungus.

Viruses and Mycoplasmas

A number of plant diseases caused by viruses and mycoplasmas may be attributed to air pollution injury by untrained persons. Virus-caused necrotic symptoms include spots, mottling, mosaics, and leaf-cupping. These symptoms should not be a source of confusion. However, tobacco mosaic virus and tobacco etch virus may cause symptoms in tobacco that mimic those of O_3.

One of the most difficult problems in distinguishing between air pollutant and virus- or mycoplasma-caused disorders is the general lack of knowledge concerning the host-range of most viruses and the symptoms they may induce on various minor crop and weed species.

Bacteria

Bacterial diseases such as halo blight of beans, wildfire of tobacco, wilt of cucurbits, and leaf infections caused by bacteria such as *Pseudomonas syringae* have symptom patterns that have been well described. Isolation of the suspected pathogen would eliminate confusion if any exists.

Nematodes

The effects of nematodes on plants are almost identical to the symptoms resulting from fungus infections of the root system. As with bacteria, isolation procedures will assist in eliminating or implicating these organisms as a cause of the symptom.

Insects

Sucking insects such as mites, thrips, leafhopping scale, and aphids that feed on leaf and stem fluids may produce symptoms similar to those produced by air pollutants such as O_3, SO_2, and F. The symptoms persist long after the feeding activity has ceased, and the insect causing the injury may therefore not be present to assist in diagnosis although remnants or frass may be present. General leaf mottle and chlorosis, or local necrotic spots, are typical symptoms of the feeding activities of sucking insects (Plate 8-11). Poor overall plant vigor with associated dieback and even death may occur following heavy infestation (Plate 8-12). The presence of insect carcasses, eggs, exuviae, frass, and microscopic examination of damaged areas will assist in accurate diagnosis.

Examples of such insect injury include mite injury on tomatoes (Plate 8-13) and soybeans (Plate 8-14). Thrip injury on grass (Plate 8-11), and lacewing injury on plants such as azalea and dogwood (Plate 8-15) also resemble ozone injury. Scale insects on eastern white pine cause a needle-browning that, from a distance, resembles emergence tipburn (Plate 8-16). However, in many of the above mentioned cases, signs of the insects' presence is noticeable with a hand lens. Examination of infested leaf material may reveal the insect itself or insect remains, webbing, eggs, or excrement. Also, insect injury on leaf surfaces tends to be more restricted than does ozone injury. For example, early red spider mite injury on beans is often seen as small patches of symptoms, perhaps 1/2-inch across, whereas ozone injury on beans may encompass the entire leaf surface. Certain insecticides, such as Kelthane, have also been reported to cause air-pollution-like symptoms (Plate 8-9).

Teratogenic Agents

Genetic Disorders

Morphological and pigmentation types of genetically-induced disorders can easily be confused with chronic exposure to various air pollutants. Recent studies with scotch pines have identified levels of green (or yellow) coloration to be associated with cultivars of this species. Such coloration appears to be similar to that caused by chronic exposure to O_3.

Summary

The air pollution diagnostician must use extreme care in separating the real air pollution-caused injury from that caused and/or aggravated by other possible causes. A hypothetical example may be the best method to illustrate and summarize the important points presented throughout this chapter.

Assume that a source of fairly high concentration of SO_2 exists near a Christmas tree plantation, a forested area, and an agricultural area, as well as being situated at the edge of an urban subdivision with well-established and also recently constructed homes. The typical SO_2 symptoms that have occurred on nearby corn plants may indeed have been induced by the pollutant, and subsequent damage claims may be sought from the industry involved. However, disorders affecting other plants in the area should not be assumed caused by the SO_2. There may be needlecast on conifers and root injury on the large white oaks near newly constructed homes, salt injury to roadside trees, drought injury from compacted soil, etc.

In this example, the diagnostician involved in determining the extent of damage caused by SO_2 should perform a comprehensive survey of the area because of the obvious other causes of plant diseases. Consideration of all factors involved and accurate and detailed notes will assist in evaluating that damage caused by SO_2 and that caused by other agents. Table 8-2 should alert the diagnostician to possible causes other than air pollutants.

Table 8-2

Cross-Reference of Symptoms Which May Be Confused for Air Pollution Injury by the Layman

If Symptoms are Similar to Those Caused by:	*Check These Other Possible Causes*
Cl	See SO_2, O_3, and F above. Check extent of affected area.

Table 8-2 (Continued)

Cross-Reference of Symptoms Which May Be Confused for Air Pollution Injury by the Layman

If Symptoms are Similar to Those Caused by:	*Check These Other Possible Causes*
F	De-icing salts; moisture stress; frost; virus infection; pesticides with oil carriers; Mn, Zn, K, N deficiency.
NH_3	Moisture stress; frost.
O_3	Virus infection; insects; frost; pesticides; Mn excess; N, P, Mg, B, Fe deficiency; moisture stress; leaf diseases caused by other organisms.
PAN	Herbicides; cold injury.
SO_2	Moisture stress; hjgh temperature; frost; de-icing salts; N, K, Mn, Mg, Ca deficiency; pesticides; some leaf diseases.

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Plate 8-1. *Heat Injury to Roadside Trees Caused by Street Paving*

Plate 8-2. *Winter Injury to Boxwood and Deodora Cedar. Many Different Agents Could Cause Similar Damage*

Plate 8-3. *Roadside Maples Affected by De-Icing Salts. Position of the Trees and the History of Salt Application are Keys to Proper Diagnosis*

Plate 8-4. *Roadside Shrubs Affected by Salt Spray from Passing Vehicles Following Use of De-Icing Salts*

Plate 8-5. *Injury to Oaks Caused by Grade Changes in Landscaping. Bronzing of Foliage and Leaf Drop Preceded Death*

Plate 8-6. *Frost Injury to White Oak. Note Similarity of Damage Symptoms to SO_2- or F-Induced Injury*

Plate 8-7. *Nitrogen Deficient Corn Leaves Showing Symptoms That Could Readily Be Confirmed Through a Soil or Foliage Test*

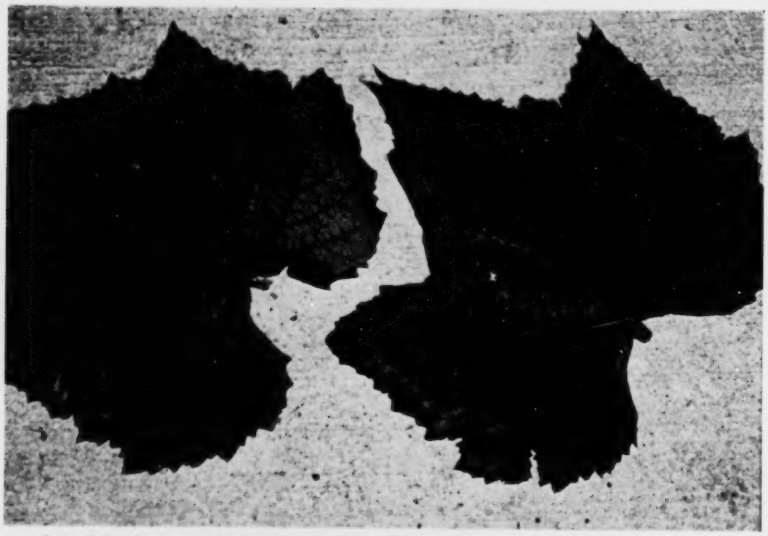


Plate 8-8. *Manganese Deficient Grape Leaves Showing Symptoms Similar to Those Induced by Ozone*

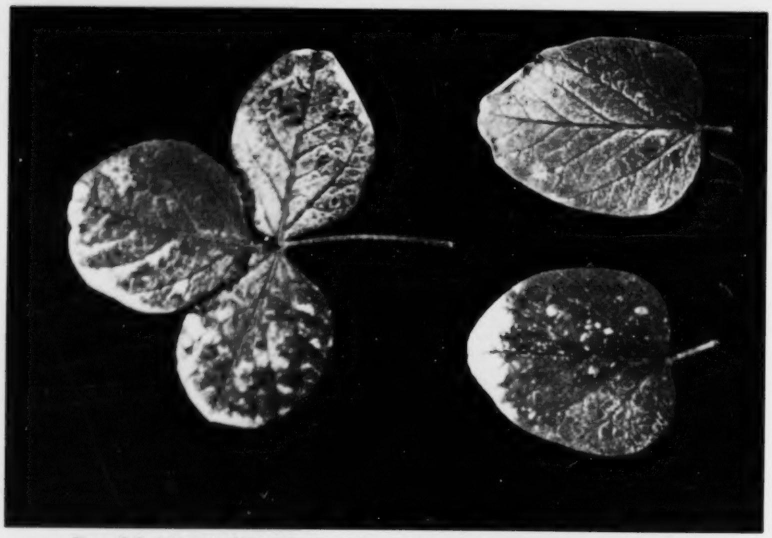


Plate 8-9. *Miticide (Kelthane) Injury on Soybean Leaves. Note Similarity to Air Pollution Injury*

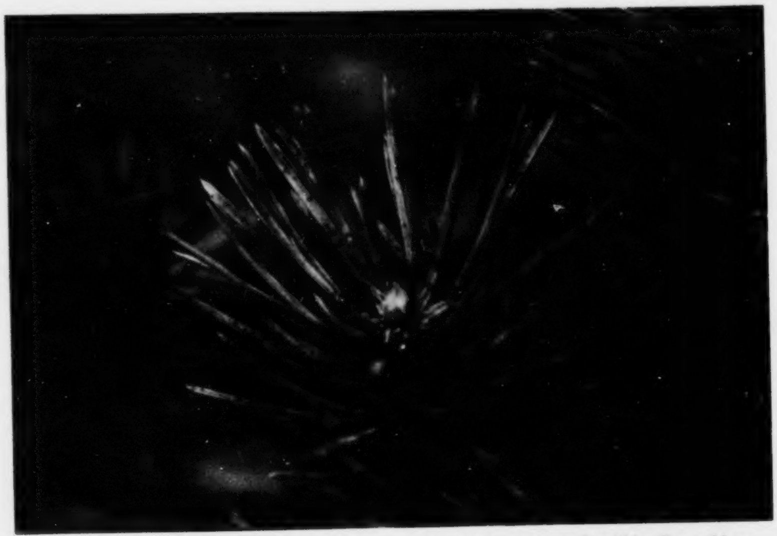


Plate 8-10. *Needlecast of Scotch Pine. Similar Initial Symptoms Could be Caused by SO_2, O_3, or F*

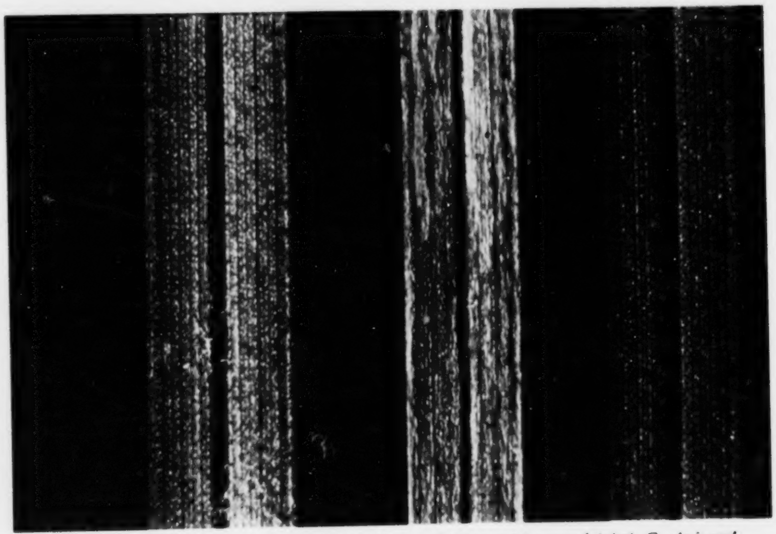


Plate 8-11. *Bluegrass Injured by Thrips (center). Normal Leaves (right), O_3-Injured Leaves (left)*

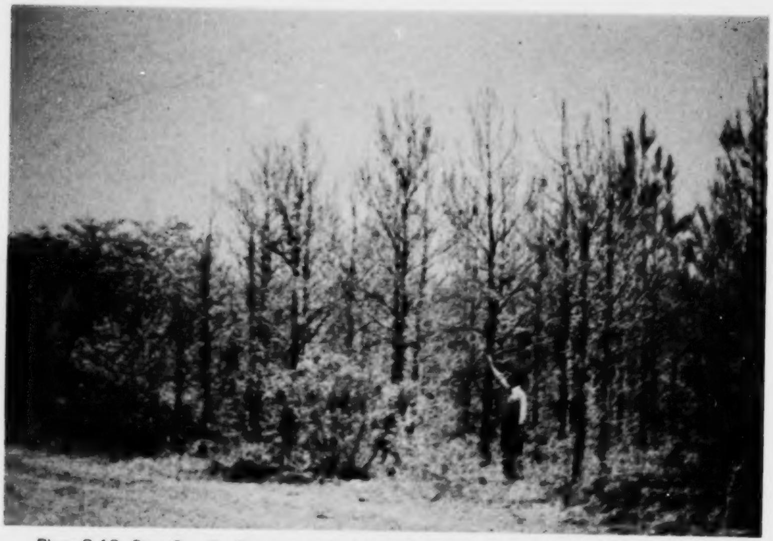


Plate 8-12. *Pine Sawfly Damage to Loblolly Pine Stand. Close Examination Would Easily Indicate This Cause Unless the Insect Was Not Present*

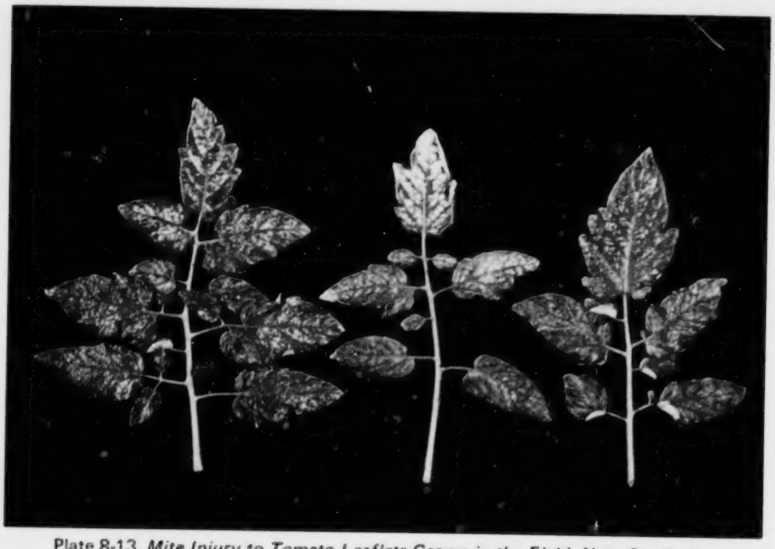


Plate 8-13. *Mite Injury to Tomato Leaflets Grown in the Field. Note Similarity to Ozone Injury*

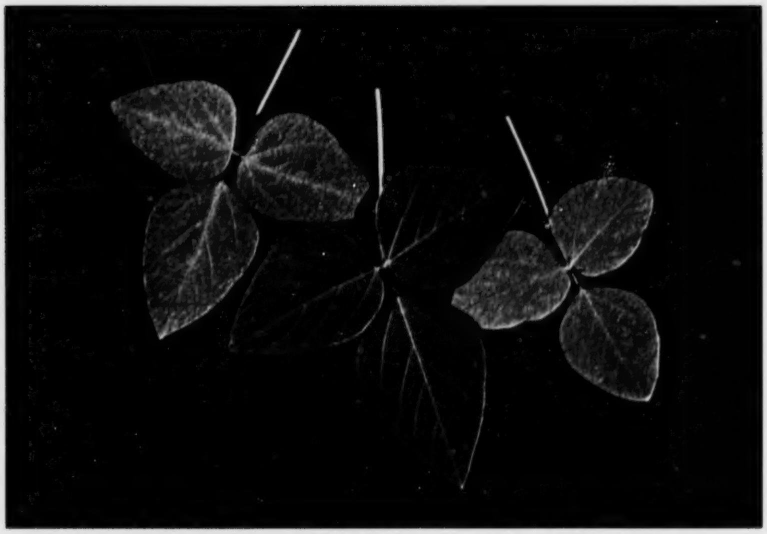


Plate 8-14. *Soybean Leaves. Left, Oxidant Injury (Probably Ozone); Center, Normal Leaflet; Right, Mite Injury*

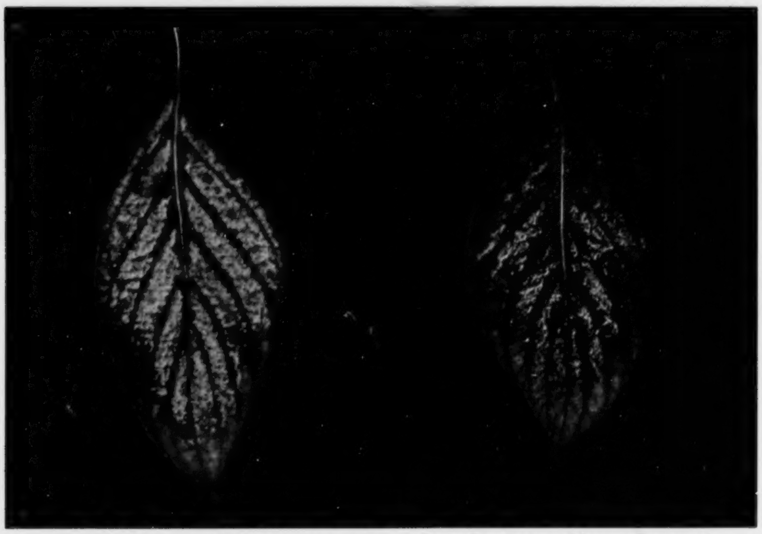


Plate 8-15. *Dogwood with Lacewing Injury. Note Similarity of Insect Damage to Air Pollution Injury*

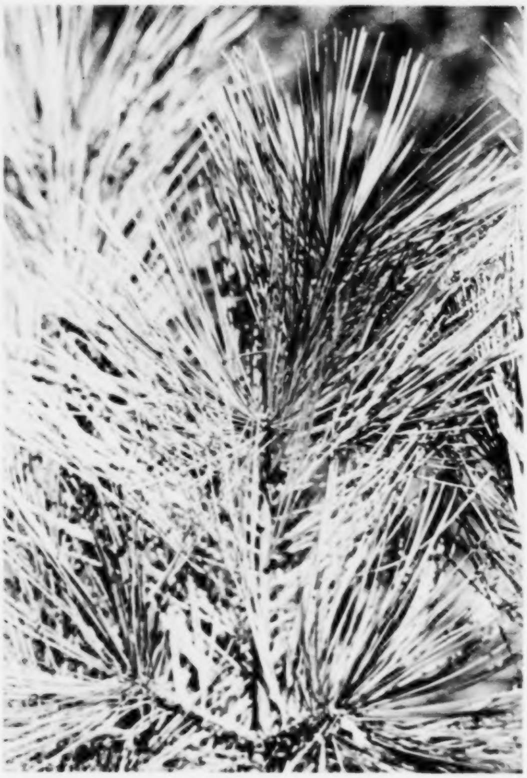


Plate 8-16. *Eastern White Pine with Oyster Scale Insect Injury. Note Similarity to Air Pollution Injury*

Chapter 9

METEOROLOGY AND AIR POLLUTION INJURY TO VEGETATION

The atmosphere is the medium in which air pollutants are emitted and transported from the source to the receptor. Although this sounds simple on the surface, it is perhaps the most complex and least understood facet of air pollution. Many variables influence the character of a given chemical species from the time it leaves the source until it reaches the receptor. A few examples will suffice to illustrate the complexity of the situation.

First, consider emission. Pollutants can be emitted from a point source such as a power plant, a line source such as a highway, or an area source such as a city or large industrial complex. The emission point may be close to the ground (e.g., the tailpipe of a car) or over a thousand feet in height (e.g., high stacks of a power plant). Thus, elevation alone has a tremendous influence on how rapidly the pollutant will be dispersed and diluted before it reaches a receptor. The relative size of the pollution source is an obvious variable. Time of emission is important because meteorological conditions vary throughout the day. As will be seen in this chapter, the atmosphere is more stable at night, and less dilution occurs then. During the day, sunlight plays an important part in transforming the chemical species of pollutants.

Second, consider the transport phenomenon. Many attempts have been made to characterize the vertical and horizontal dispersion of pollution from point and line sources. Many mathematical equations and models have been developed. Each has deficiencies because of the variability of sources, source strength, topography, and other factors. From the receptor standpoint, this is the important phase because, if the pollutants are not adequately dispersed and diluted, atmospheric insults will occur.

There are many factors to consider and it should be kept in mind that a nearby source does not necessarily imply that damage will result.

This chapter will attempt to point out the salient points about the relationship between meteorology and air pollution and the effect of weather on pollutant dispersion.

Sources of Air Pollution

The sources of pollution have been well characterized over the past few years, and much public attention has been focused on the topic. These sources can be briefly categorized into four groups: industrial, urban, mobile, and rural.

Industrial Sources

Industrial operations emit many different types of pollutants, depending upon the type of industry. One area of information that can be useful in the pinpointing of an industrial source is the emission inventory developed by the local air pollution control agency. These inventories can be helpful in determining which industries in a given area are likely to be emitting a suspected pollutant. However, in many industrialized areas, several industries may emit the same type of pollutant. This can make pinpointing a specific source impossible. Nevertheless, emission inventories can still serve as a valuable tool in identifying the pollutant (or the absence of a pollutant) and probable industrial source(s) of air pollutants.

Urban Sources

This group includes commercial incinerators and small furnaces used for domestic heating purposes. Individually, these sources emit far smaller amounts of pollutants than industrial sources, but because of the large number in a given urban area, the sum total of their emissions has become a significant source of air pollutants.

Mobile Sources

Mobile sources, of course, include the automobile. This category is responsible for several pollutants: carbon monoxide, hydrocarbons, and nitrogen oxides. Nitrogen oxides and hydrocarbons are the most important of these, since they are involved in the formation of ozone and peroxyacetyl nitrate, both of which are major causes of plant injury. This source is easy to identify, but it is impossible to pinpoint a specific polluter responsible for damage because of the large number of vehicles contributing pollutants to the atmosphere.

Rural Sources

This group includes pollutants from field spraying of chemicals, agricultural burning, forest fires, etc. Most of these are isolated sources that tend to be seasonal.

Meteorological Aspects of Air Pollution

Given the emission of a pollutant from one of the above-named sources, the next step in the chain of events leading to pollution injury is *transport* and *dispersion* of the pollutant from the source to the receptor. The major meteorological factors influencing transport and dispersion of air pollution are: (1) wind speed and direction, and (2) atmospheric stability and the temperature.

Wind Speed and Direction

This is easily understood, since it determines the predominant direction in which the pollutants will move and the speed at which they will be transported and diluted downwind. Both speed and direction are concerned mostly with horizontal movement of the air containing the pollutants. When referring to wind direction, the direction from which the wind is blowing is the name given to that wind. Hence, a northwest wind is one blowing from the northwest to the southeast. The speed of the wind can often influence the toxicity of certain pollutants by determining the amount of time the pollutants spend in the atmosphere undergoing chemical changes. Such changes can cause some pollutants to become more toxic and others to become less toxic. In addition, the speed of the wind is one factor that determines the volume of air that passes by the source. The higher the wind speed, the greater the dilution of the effluent, other factors remaining constant. Thus, the dilution of a pollutant, once it is emitted into the atmosphere, is directly proportional to the wind speed. Data on wind direction and speed can be used to eliminate possible sources that are downwind of the damage site at the time of the pollution episode or are too distant either upwind or downwind to be considered probable sources, given the wind speed at the time. Thus, appropriate sources upwind of an affected area can be studied more thoroughly to assess their impact.

Atmospheric Stability and Temperature

The stability of the atmosphere, as an index of turbulence, can be related to the vertical mixing of pollutants. A good deal of the atmospheric turbulence is associated with thermal turbulence, in which mixing is enhanced due to temperature variations between different altitudes.

Lapse Rates. On a clear day, when the sun is shining, the sun warms the surface of the earth very effectively. This heat is then transferred by conduction and radiation to the atmosphere near the ground, increasing the air temperature as a result. This warmer, less dense air then rises and the cooler, denser air of the upper atmosphere descends. Any pollutants emitted into the air under these conditions are caught in this vertical mixing and dispersed rather rapidly. Unfortunately, these conditions do not always exist.

Under normal, dry conditions the temperature of the air decreases at the rate of approximately 1°C per 100 meters (5.4°F per 1,000 feet) of altitude. This decrease in temperature with increasing altitude is termed "the dry adiabatic lapse rate." The dry adiabatic lapse rate is compared with the "environmental lapse rate" (current or actual temperature change with altitude) to yield an index of stability. Air is said to be ***stable*** whenever the vertical decrease in temperature – the environmental lapse rate – is less than 1°C per 100 m, and *unstable* when the environmental lapse rate exceeds 1°C per 100 m. As shown in Figure 9-1, it follows that an unstable condition, the environmental lapse rate with the least slope, represented by the line to the right of the adiabatic lapse rate line, increases vertical mixing, and pollutants emitted under this condition are dispersed relatively quickly. Conversely, pollutants emitted into a stable atmosphere, represented by the line to the left of the adiabatic lapse rate line, are not dispersed as quickly, due to decreased vertical mixing.

One additional condition exists when the temperature increases rather than decreases with altitude (Figure 9-2). When the temperature of the air at higher altitudes is warmer than that near ground level, a "temperature inversion" is said to occur. An inversion condition tends to restrict vertical mixing, keeping the pollutants trapped in the inversion layer since cooler, denser air is now below a warm, less dense layer of air.

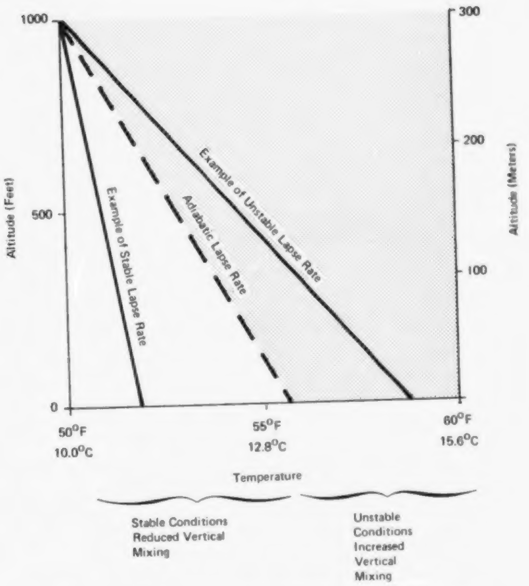


Figure 9-1. *Temperature Variations with Altitude*

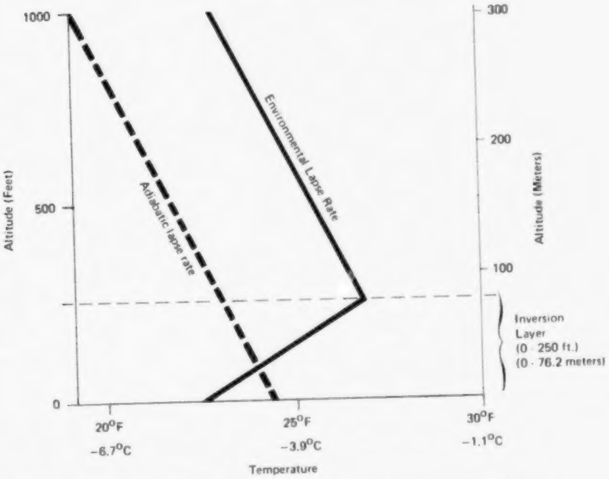


Figure 9-2. *Temperature Variation with Altitude Illustrating a Temperature Inversion Based at the Earth's Surface*

Figure 9-3 illustrates a set of conditions in which the altitude of an inversion plays an important role in the concentration of air pollutants. In the example given, there are two inversion layers present: one from ground level to approximately 250 feet (called a surface-based inversion) and one from about 900 to 1,000 feet (called an inversion aloft). Figure 9-4 illustrates the conditions in Figure 9-3 so that the effects of these conditions on the dispersion of pollutants can be determined more easily. As can be seen, the emissions from the homes and small industry on the right are being trapped and concentrated beneath the surface-based inversion, since vertical mixing is restricted. The taller industrial stacks, however, are emitting their pollutants above the surface-based inversion and, as a result, the pollutants may be fairly well mixed, depending on the wind speed in the layer from 250 to 900 feet. Remember, however, that the amount of pollutants emitted from tall stacks remains the same and that adverse effects may be noted at greater distances downwind.

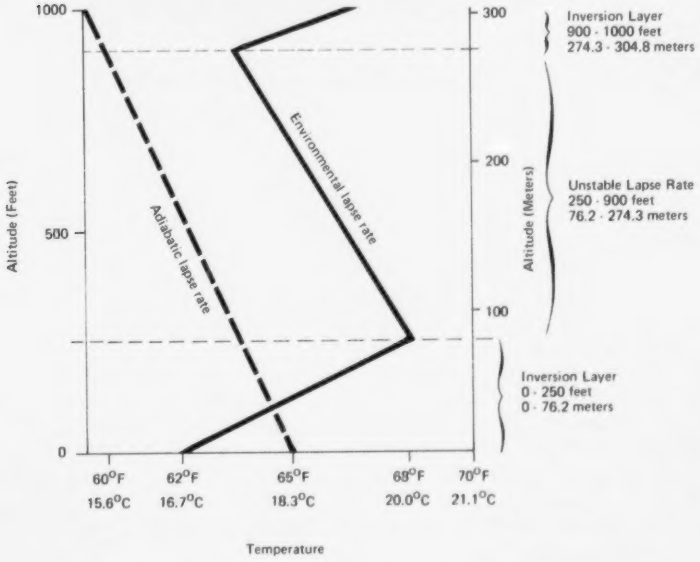


Figure 9-3. *Temperature Profile Illustrating Two Inversion Layers*

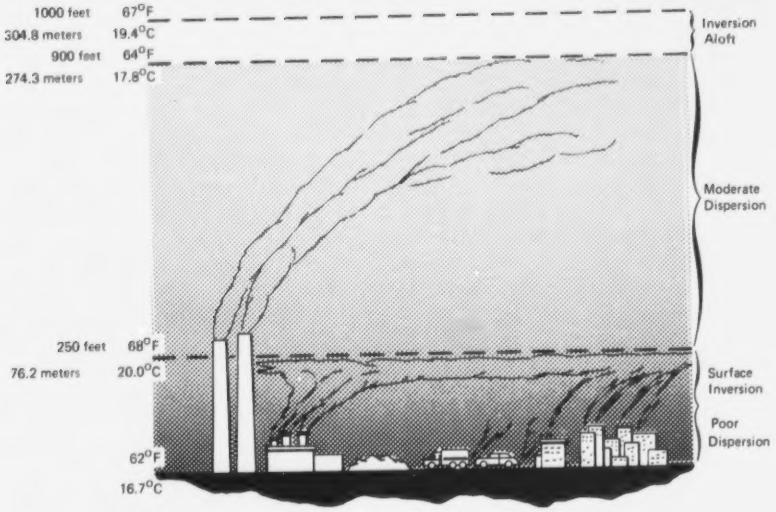


Figure 9-4. ***Conditions of Figure 9-3 in a Different Perspective***

Diurnal Temperature Variations. Another factor that has an effect on vegetation injury due to air pollution is the diurnal (daily) cycle of temperature variation through the atmosphere. A typical daily cycle of temperature gradients over open country on a cloudless day begins a few hours after sunrise with the formation of an unstable lapse rate at the ground. The warm layer increases in height during daytime due to strong solar heating of the ground. This is associated with a high degree of vertical mixing.

Shortly before sunset, the air near the ground begins to cool rapidly and a stable lapse rate begins to develop. During the early evening an inversion forms, reaching maximum intensity and height near the time of minimum surface temperature. During this period, pollutants are trapped within the inversion layer and accumulate as a result of decreased vertical mixing coupled with low wind speeds. At this point pollutants emitted at the surface are not carried aloft, while pollutants emitted aloft, for example, by tall chimneys, are not generally transported to the ground.

With the coming of daylight, the ground begins to warm and the inversion is destroyed. This may result in "fumigations;" that is, the rapid downward mixing of pollutants that were released aloft during the night. This will often lead to high pollutant concentrations during the early forenoon before normal, vigorous daytime vertical mixing is resumed. The high concentration of pollutants at ground level during the forenoon may coincide with the opening of the stomata of plants. As a result, this period becomes a prime time in which vegetation injury may occur.

Diagnostic Confusion Caused by Meteorological Conditions

Temperature, humidity, and precipitation are meteorological conditions that can sometimes confuse diagnosis of injury. Extremes in temperature or precipitation can produce visible symptoms similar to and often indistinguishable from air pollution injury. On the basis of information on these variables, they may be either included as possible causes of the observed symptoms or excluded if the conditions were near normal at the time.

Effects of Topography on Dispersion

Finally, another factor – the local topography of an area – can interact with meteorological variables to affect the dispersion of pollutants. Valleys can become pockets from which pollutants cannot escape, especially under stable conditions. The pollutants can be channeled along the valley, perhaps affecting areas many miles removed from the source, or they may simply stagnate in a sheltered area for a prolonged period of time. Mountains can act as a barrier over which pollutant-laden air cannot flow, thereby resulting in a buildup of the pollutants. An example of this is the Los Angeles area, where the nearby mountains form a barrier, and frequent temperature inversions above the surface allow "smog" to accumulate. On a much smaller scale, a row of trees or buildings can act as either a barrier or a channel for air pollution. Clearly, local terrain must be taken into account when attempting to map the route of pollutants back to their source.

Meteorological data for a given period of time can be obtained from the U.S. National Weather Service Offices. Records of local observations may also be available from local airports, yacht clubs, agricultural stations, universities, and individuals or companies. All available information, including a description of the local topography, should be examined in order to increase the accuracy of tracing a suspected pollutant to its likely source.

Chapter 10

DIAGNOSIS OF SUSPECTED AIR POLLUTION INJURY TO VEGETATION

Diagnosis of the nature and causes of suspected air pollution injury to vegetation is usually a complicated process. The factors governing plant growth are complex and interrelated, and an observer must evaluate as many of them as possible when making a diagnosis. He must consider all the symptoms that are present, separate relevant from irrelevant data, and determine the role of each possible causal factor. The observer should understand the basic procedures of diagnosis, know what background information to seek, and understand how to interpret information.

Diagnosis – Basic Procedure

The following logical sequence of seven questions forms the basis of the diagnosis of suspected air pollution injury to vegetation:

1. Is there a pollution source nearby capable of causing injury?
2. What are the characteristics of the terrain or location?
3. What are the symptoms of injury and what part of the plant is affected?
4. What is the distribution of affected plants?
5. Are biological agents present?
6. Is more than one species of plant affected?
7. What is the crop history of the affected area?

1. Is There a Pollution Source Nearby Capable of Causing Injury?

The obvious question to ask when air pollution is suspect is whether or not a source is within range of the affected vegetation. Nothing is served by insisting that the observed symptoms were caused by a given pollutant if it can be determined that no nearby source exists. On the other hand, one must keep in mind that long distance transport of certain pollutants is possible. Chapter 3 discusses transport of oxidant from the Los Angeles basin to the San Bernardino mountains some 50 miles distant. Dispersion equations for point, area, and line sources of pollution are available to determine ground concentrations at various distances from the source.

2. What Are the Characteristics of the Terrain or Location?

Terrain characteristics of both the affected area and the area around the source, and the location of affected plants in relation to terrain are important factors to consider when diagnosing suspected air pollution injury. Terrain influences the dispersion of an air pollutant and, consequently, the geographic distribution of affected plants.

The local terrain may prevent dispersion of the air pollution if a point source of air pollution is located in a steep, narrow valley, and vegetation damage will be concentrated on the floor and sides of the valley. Air pollutants are also trapped by large terrain features, such as the horseshoe-shaped Los Angeles basin, which prevents the dispersion of air pollutants over a wide area.

The location of plants in relation to local terrain features is another important factor to consider in diagnosis. For example, plants located in a low area in a field may receive excessive water during wet periods. The excess water may drown the roots, leach out nutrients, or create conditions that favor disease development. The low area may be a frost pocket in the spring or early fall.

Soil conditions also vary within a local area and must be considered when diagnosing plant ailments. The type of soil may be quite different in a low area compared with a nearby high spot. Soil or plant tissue analyses for mineral deficiencies are a useful aid to correct diagnosis.

Location is important in other types of injury that may be confused with air pollution injury. Plants growing in exposed locations, such as at the corner of a house or on an open golf course, may exhibit severe winter injury while nearby protected plants remain uninjured.

3. What Are the Symptoms of Injury and What Part of the Plant is Affected?

A symptom is a visible change in a plant caused by some physiological abnormality or stress. Although symptoms may appear on any portion of the plant, those associated with air pollution injury usually appear on the foliage.

One of the first steps in diagnosing suspected air pollution injury to plants is to closely examine the foliage. The color of the injured tissue, pattern of injury on the leaf, which leaf surface is affected, state of maturity of the leaf, and other such conditions should be noted. Foliage that has fallen to the ground should also be examined. Both detailed field notes describing the symptoms and photographs are extremely useful, especially if samples are collected to be keyed out later. For comparison purposes, healthy foliage from plants growing in adjacent areas should be examined.

Acute symptoms may appear within 24 hours after an air pollution episode. Few other factors of the environment cause such rapid injury and symptom development. Consequently, many factors can be eliminated as possible causal agents based solely on this time factor.

The length of time that passes between symptom development and observation is also important. Leaves with classic air pollution symptoms may fall or wither on the plant over a period of several weeks, precluding an accurate diagnosis. Also, new growth may hide remaining symptomatic leaves, especially on larger plants such as trees. Decay fungi and insects may cause additional damage to injured leaves, thus distorting or obliterating symptoms of air pollution damage. Therefore, it should be remembered that the final symptom on a plant leaf is the end result of a series of complex ecological and physiological relationships. The longer the time period that occurs between symptom development and observation, the more complex the diagnosis becomes. Whenever possible, symptoms should be evaluated as soon as they are manifested and followed through their development if possible.

Although the specific symptom evaluation is very important, it must be considered in context with many other factors of the evaluation to get the proper perspective of the overall problem. Symptom description must be combined with a knowledge of tissues and organs affected, species of plants involved, time of symptom appearance, and geographic distribution of the problem.

When air pollution injury is suspected, it should be determined if the entire plant is affected or if only certain stems or branches show symptoms. Air pollution will often injure a plant rather uniformly, although certain aged leaves or tissues may be more severely injured than others. If only one stem or branch of a plant shows injury, the base of the injured portion should be examined for a canker, break, or other injury that could cause the entire stem or limb to die from that point outward. However, it must be remembered that problems such as root or basal stem cankers, soil nutrient deficiencies, or water imbalances will often also affect the aboveground portion of a plant rather uniformly.

The approximate age of affected leaves may be of importance. For example, whereas few air pollutants injure the extremely young, immature leaves, recently matured leaves may be very sensitive to air pollution injury. Therefore, severe injury on young foliage just emerging in the spring may be more likely due to frost, herbicides, or something other than air pollution.

Sunscald, winter injury, or wind injury might be suspected if the injury is predominantly on one side of a plant. Only a very local, and probably readily identifiable, point source of air pollution would cause such injury on the side of the plant facing the source.

4. What is the Distribution of Affected Plants?

The distribution of affected plants, together with the symptomatology, will often yield a correct diagnosis when dealing with suspected air pollution injury around a point source. Damage is usually most severe downwind from the source and becomes less severe with increasing distance from the source. Less injury is observed upwind or at crosswind locations with respect to the source. The pattern or distribution of affected plants will vary according to terrain, meteorology, and specific source features such as stack height.

In the case of a general, widespread source of air pollution, such as a large urban area, distribution of affected plants will be widespread in and adjacent to the urban area. Pollutants may drift away from urban areas and cause injury to vegetation growing in rural areas miles away. As with a point source, damage is normally most severe downwind from the urban area.

There are often both tolerant and sensitive individuals within a population of plant species. A plant injured by air pollution may be growing next to an uninjured plant of the same species. This confusing situation is more often the rule than not and should be remembered when determining the distribution of affected plants.

If symptomatic plants are located in a very small area and no local source of air pollution is nearby, problems directly associated with the affected area should be investigated. For example, if the area is in a housing development, yard care practices involving the use of pesticides, herbicides, fertilizers, or other chemicals should be examined. If the area is rural, natural features such as high water tables, poor soil, or frost pockets should be considered as possible causes of injury. In addition, if the area is farmed, overfertilization, herbicide spills, or other accidents must also be investigated.

5. Are Biological Agents Present?

Many biological agents cause symptoms on leaves that mimic those caused by air pollution. At times, these agents are easily recognized and diagnosis is fairly simple. The causal organism or its remains may be present on the leaf. For example, mite injury on broad-leaved plants closely resembles ozone injury. However, close examination of affected tissue with a hand lens may reveal the mites, their webs, or the skeletons of dead pests. Also, the specific tissue attacked by mites or insects may be quite different from that injured by air pollution.

If a fungus is involved, diagnosis may be more complex. Fungi exist in or on the leaf as microscopic, thread-like filaments. Occasionally, fungal fruiting structures are visible as small, dark bumps on the leaf surface, but the mere presence of fungi on an injured plant does not prove that the injury is fungi-induced. Many fungi normally inhabit leaves, often sporulating on dead leaf tissue killed by various other agents. Isolation and correct identification of fungi, viruses, or bacteria as causal agents of leaf injury are best performed by experienced persons.

6. Is More Than One Species of Plant Affected?

Insects, fungi, and other biotic agents are often quite selective and attack only certain plant species. In contrast, air pollutants often injure a wide spectrum of plants, especially in the case of a severe fumigation. Thus, determining how many plant species exhibit injury can help an observer distinguish air pollution injury from other kinds of injury.

Grasses, herbs, deciduous trees, and conifers may all show air pollution injury to various degrees. Highly sensitive plants will be most severely injured, while more tolerant plants will show few, if any, symptoms. Symptoms may appear on only the most sensitive species if the air pollution episode is not severe. A combined knowledge of the classic symptom pattern and the sensitivity of various plant species is most helpful in diagnosis. It is important to examine the highly sensitive "indicator plants" for presence of symptoms, but it is equally important to examine species known to be tolerant to the suspected air pollutant to confirm the lack of symptoms.

Other agents of the environment in addition to air pollution injure a wide spectrum of plant species. Frost, drought, flooding, and other natural environmental extremes are examples. Man-made factors such as herbicides, excess fertilizer, and improperly-used pesticides may also damage numerous species. However, the symptoms and other considerations of the overall syndrome often aid in separating air pollution injury from other kinds of injury.

7. What is the Crop History of the Affected Area?

The symptoms observed when making a diagnosis may actually have been caused or influenced several weeks earlier. Thus, the crop history prior to and following a suspected air pollution episode must be carefully considered when making a diagnosis. For example, if the injury is observed on farm crops, management practices such as crop rotation should be investigated. Information should be gathered on the type and treatment of crops grown in the soil prior to the present problem. Certain crop plants do not grow well following certain other crops in the same soil. Continual cropping of the same species may also cause various problems.

Most farmers keep good records on crop rotation, amount of fertilizer and other agricultural chemicals applied, etc., and some keep records on yield. Many farms have farm plans prepared by the Soil Conservation Service to prevent soil erosion, and a fairly rigid crop rotation will be followed on those farms. However, there are farmers that keep very few or no records. Also, one must expect that answers to questions about over-application of pesticides may not always be candid.

The amount and type of fertilizers, pesticides, and herbicides used in the past should be determined. Certain combinations of these chemicals are incompatible and may cause severe plant damage resembling air pollution injury. Exceeding recommended dosages likewise may cause severe injury. Some chemicals remain in the soil and build up for years to toxic levels.

Pesticides applied to nearby farmlands or to adjacent yards should be investigated as well. Certain herbicides can drift for long distances and cause injury to sensitive plants. For example, application of weed killer in a yard, or brush killer in a right-of-way, may injure sensitive plants growing as far as half a mile away.

Diagnosis – Systematic Approach

These seven questions are the basic elements of a proper diagnostic method for suspected air pollution injury to vegetation. Not only must these questions be answered, but also they must be approached in a systematic fashion. Once an observer trains himself to deal with these questions in an orderly fashion, diagnosis will become a second-nature routine that will come automatically when confronting a field problem.

One of the obstacles impeding the accurate diagnosis of air pollution injury has been the failure of many investigators to utilize a systematic method of gathering and analyzing the information about an episode. Sometimes investigators rely too heavily on their past experiences to determine the likely causal factor. As a result, they have often reached their conclusion before considering all the pertinent information available to them. In other words, they have sometimes allowed their individual biases to lead them to an erroneous diagnosis of the problem. The remainder of this chapter describes a systematic step-by-step method of obtaining and handling the pertinent information in such a way as to increase the probability of reaching a correct diagnosis of the cause of plant injury.

Hypothetical Example

For the purpose of introducing a systematic method of gathering and analyzing information concerning plant injury, a simple, hypothetical example of an incident that appears to be air pollution-related is presented below. It will demonstrate that meteorological data, information from emission inventories, and information on the relative sensitivities of different plants to different air pollutants are often sufficient to determine the likely causal pollutant, if any, and its probable source.

Diagnosis of actual instances of suspected air pollution injury to vegetation is normally more complex than this example suggests for two reasons. First, actual sensitivity ratings are not as exact as they appear on the chart, because researchers have differing opinions about sensitivity and because different cultivars of plants listed often differ in sensitivity to a pollutant. Second, the sources in the example can also be emitting pollutants other than the ones listed. For example, the city and the smelter could both be adding SO_2 in addition to the pollutants listed. The field diagnosis of plant injury thus relies heavily on the discrimination of the visible symptoms of injury. However, the combination of an accurate discrimination of symptoms with a logical approach of assessing the meteorological data and source information will often lead to an accurate diagnosis of the problem.

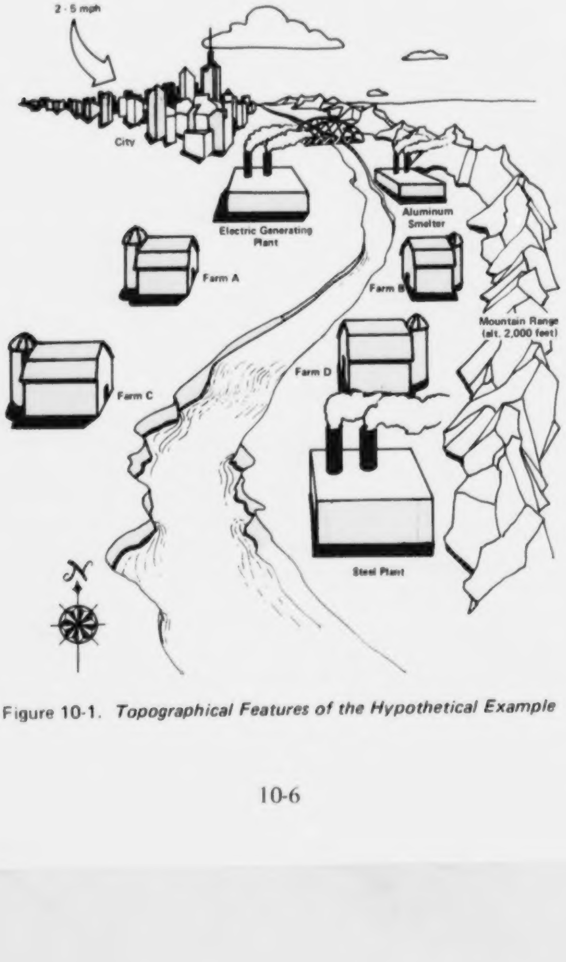


Figure 10-1. *Topographical Features of the Hypothetical Example*

Consider the following example of injury to some vegetation in the area depicted in Figure 10-1. The following information is needed to deduce the causal pollutant, if any, and its probable source.

1. Symptoms observed:
   a. Farm A – Wheat injured
      Cucumber not injured
   b. Farm B – Oats injured
      Onion not injured
   c. Farm C – Eastern white pine trees injured
      Corn not injured
      Chrysanthemum not injured
   d. Farm D – Alfalfa injured
2. Meteorological conditions in the city at probable time of plant injury:
   a. Wind from northwest at 2 to 5 mph
   b. Inversion layer base at 500 feet
   c. Sun shining
3. Possible sources:
   a. Smelter – possible source of fluoride (F)
   b. Generating plant – possible source of sulfur dioxide (SO_2)
   c. Steel mill – possible source of sulfur dioxide (SO_2)
   d. City – possible source of ozone (O_3), PAN, carbon monoxide (CO)
4. Topography – Mountain range with farms, factories, and city near river in valley to west. See Figure 10-1.

Given the above information, what pollutant, if any, probably caused the injury, and what is the likely source of the pollutant? The following is a step-by-step solution of the problem.

(1) Sensitivity charts reveal:

	Pollutants*			
	F	SO_2	O_3	PAN
Wheat	T	S	S	I
Cucumber	T	T	T	T
Oats	I	S	S	S
Onion	+	T	S	T
Eastern White Pine	S	S	S	+
Corn	S	T	S	T
Chrysanthemum	T	T	S	T
Alfalfa	T	S	S	IS**

S = sensitive
I = intermediate
T = tolerant
+ = not rated in literature

Note: these are hypothetical ratings

*CO not known to exist in high enough concentrations in ambient air to cause injury.

**Different cultivars may vary in their sensitivity to specific pollutants.

This chart reveals the possible pollutants based on observed injury to the plants:

a. Wheat was injured and is sensitive to SO_2 and O_3. (This tends to eliminate F and PAN.)

b. Cucumber was not injured and is tolerant to SO_2, F, O_3, and PAN. (Thus, SO_2 and O_3 remain suspect, while F or PAN injury becomes more unlikely.)

c. Oats were injured and are rated sensitive to SO_2, O_3, and PAN and tolerant to F. (No change in suspicion.)

d. Onion was not injured and is sensitive to O_3 and tolerant to SO_2 and PAN. (Now only SO_2 remains suspect. If O_3 were the cause, the onion probably would have exhibited symptoms.)

Continuing down the list of injured plants further supports the conclusion that SO_2 was the likely causal pollutant.

(2) A survey of possible sources reveals:
   a. Smelter – eliminated (not a source of SO_2)
   b. Generating plant – possible
   c. Steel mill – possible
   d. City – eliminated (not a source of SO_2)

(3) The meteorological and topographical factors reveal:
   a. A buildup of pollutants caused by the low inversion layer and mountains to the east, which prevent dispersion.
   b. A low wind speed from the northwest, further restricting dispersion and eliminating the steel mill as a possible source.

(4) Final conclusion:
   a. Probable causal pollutant – SO_2
   b. Probable source of pollutant – generating plant

Diagnostic Routine

In order to structure a logical approach to the analysis of the pertinent information about suspected air pollution injury, the remainder of this chapter presents a step-by-step routine to assist in the diagnosis of the cause of plant injury. The routine utilizes a branching procedure that leads through a series of steps to a final conclusion for a particular set of circumstances. Before presenting the routine, however, it is advantageous to describe the characteristics of the branching procedure.

Each step of the procedure contains a description of what to look for, or some other discussion, and is usually followed by a question that must be answered in order to be directed to the next step. This question will almost always list all the alternatives from which to select the appropriate answer. After selecting the answer, the next step in the procedure is indicated. This listing of questions, alternative answers, and branching procedures, may tend to appear too long and complex to be of use, but this is definitely not the case. Figure 10-2 depicts part of a hypothetical branching procedure with one possible pathway through all the steps shaded. This represents the one (and only) correct path that leads to the conclusion to one particular problem.

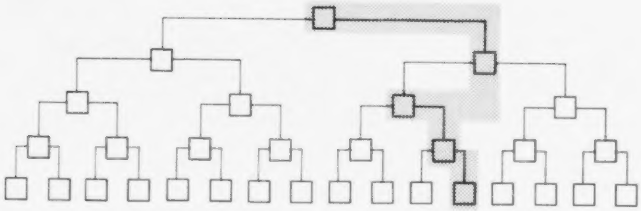


Figure 10-2. *The One Pathway Followed to Solve a Particular Problem*

Only a very small portion of the entire procedure is used for the example problem illustrated in Figure 10-2. By following this step-by-step branching procedure the unused steps would not even be encountered. In following this path, only five of the procedure's 31 steps to reach a final conclusion are used. The next time the procedure is used, the problem situation will in all likelihood be different, with a different set of circumstances, and as a result, a different pathway will be used. For this reason the procedure must include all possibilities, another factor that adds to the bulk of a branching procedure, but this increased bulk is far outweighed by the advantage that all possibilities, no matter how remotely possible, are always taken into consideration.

The diagnostic routine of this chapter makes use of a branching technique; however, for any given situation only a portion of the entire routine will be used. The routine contains 100 steps of which the shortest pathway consists of only six steps and the longest 25. The average number of steps required to reach a final conclusion is 17, and most of those require only very simple yes or no decisions.

The diagnostic routine is designed to do one of two things: (1) lead to a conclusion that the observed injury was in all likelihood caused by air pollution, in which case the routine will often indicate the causal pollutant and its source; or (2) eliminate air pollution as the cause of the injury either because of a lack of conclusive information or because of a determination that another factor is a more likely cause.

In utilizing the diagnostic routine, several things must be kept in mind. First, the scientific study of the effects of air pollution on vegetation is still a young field and more research is needed to resolve many unanswered questions. As a result, gaps in information exist and in some cases must be filled with "educated guesses." Second, plants are living things with individual differences, and they sometimes react differently to identical stimuli. This entire diagnostic routine is built on generalities which may fail when confronted with individual variations. Third, the successful completion of the entire routine depends heavily on the successful completion of each individual step. There is nothing to be gained by eliminating steps or hurriedly finishing the intermediate steps. Therefore, care must be taken to complete each step in the proper sequence and follow all directions contained within a step.

1. Observe symptoms on injured plants.

GO TO STEP 2

2. Construct a sensitivity chart containing both the affected and unaffected species. This will help in recalling the symptoms on the different species. Use the following format:

	Pollutant				
Injured Species	O_3	PAN	SO_2	F	. . .
1.					
2.		Enter in these boxes			
3.		the relative			
⋮		sensitivity			
Unaffected Species		rankings found			
1.		in Appendix C.			
2.					
3.					
⋮					

GO TO STEP 3

3. The observed injury can be attributed to one of the following causal categories.
   a. Insects
   b. Nutrient Deficiency
   c. Environmental Stress
   d. Biotic Disease
   e. Air Pollution

   The following steps in this diagnostic routine will attempt to resolve the confusion of diagnosis as an air pollution problem or one of the other possibilities.

GO TO STEP 4

4. Is there any evidence of an insect pest? Such evidence includes:
   a. the insect
   b. skeletons
   c. excrement
   d. webs
   e. eggs

No – GO TO STEP 7
Yes – GO TO STEP 5

5. Is the symptom consistent with the symptom normally associated with insect evidence present?

No – GO TO STEP 7
Yes – GO TO STEP 6

6. At this point it is reasonable to conclude that the insect is the probable cause of the injury. This does not eliminate the possibility of an air pollution problem, but at least for the present time air pollution cannot be named as a sole causal factor producing the symptoms.

STOP

7. How many different species of plants are exhibiting symptoms of injury?

One – GO TO STEP 50
More than one – GO TO STEP 8

8. Are all the affected species rated sensitive or intermediate to the same pollutant or pollutants?

No – GO TO STEP 23
Yes – GO TO STEP 9

9. Are any native plants affected?

No – GO TO STEP 29
Yes – GO TO STEP 10

10. The chance of a nutrient deficiency is so slight that it can be assumed there is not a deficiency.

GO TO STEP 11

11. Consult the local air pollution control agency to obtain information in the emission inventories concerning the sources of pollutants in the area and the pollutants they can be emitting. Remember that if one of the pollutants under consideration is a photochemical oxidant there will not be a source in the inventories.

GO TO STEP 12

12. Are there any sources in the area that could be emitting pollutants to which all the affected plants are rated sensitive or intermediate? Remember that if a photochemical oxidant is under suspicion, the source may be many miles away (refer to Chapter 3).

No – GO TO STEP 84
Yes – GO TO STEP 13

13. Obtain as much data as possible about the following meteorological variables:
    a. precipitation
    b. temperature
    c. wind direction
    d. wind speed
    e. inversions

    Collect data for the following time periods:
    a. If the injury appears to be chronic, collect as much as one year's data to determine the prevailing wind.
    b. If the injury appears to be acute, collect data from a day or two immediately preceding the onset of symptoms.
    c. If a strong possibility exists that the symptoms are environmental stress symptoms, then collect data for the month preceding the onset of symptoms.

    Such data are available from the U.S. Weather Stations, local airports, yacht clubs, Coast Guard, local air pollution control agency, agricultural experiment stations, universities, companies, and individuals.

GO TO STEP 14

14. Was either temperature or amount of precipitation extreme in either direction during the relevant period?

No – GO TO STEP 17
Yes – GO TO STEP 15

15. Are symptoms consistent with symptoms normally associated with the type of environmental stress indicated in step 14?

No – GO TO STEP 17
Yes – GO TO STEP 16

16. Environmental stress is the most likely cause of the observed injury.

STOP

17. Study the local topography of the area, noting terrain features such as mountains, valleys, etc.

GO TO STEP 18

18. Using the information collected on the meteorological conditions, topography, and the emission inventories, is there a possibility that a source(s) in the area emitted a pollutant(s) that could have reached the site of injury?

No – GO TO STEP 84
Yes – GO TO STEP 19

19. How many pollutant sources can be identified?

One – GO TO STEP 32
More than one – GO TO STEP 20

20. Are the observed symptoms specific for any of the pollutants from the sources under consideration?

No – GO TO STEP 34
Yes – GO TO STEP 21

21. How many pollutants still remain suspect?

One – GO TO STEP 35
More than one – GO TO STEP 22

22. At this point a decision as to a causal pollutant depends on the skill applied to discriminating between different pollutant symptoms. It is suggested that the first half of the handbook be consulted to help in the discrimination and also contact a plant specialist.

STOP

23. Are any native plants affected?

No – GO TO STEP 47
Yes – GO TO STEP 24

24. The chance of a nutrient deficiency is so slight that it can be assumed there is no deficiency.

GO TO STEP 25

25. Obtain as much data as possible about the following meteorological variables:
    a. precipitation
    b. temperature
    c. wind direction
    d. wind speed
    e. inversions

    Collect data for the following time periods:
    a. If the injury appears chronic, collect as much as one year's data to determine the prevailing wind.
    b. If the injury appears acute, collect data from a day or two immediately preceding the onset of symptoms.
    c. If a strong possibility exists that the symptoms are environmental stress symptoms, then collect data for the month preceding the onset of symptoms.

    Such data are available from the U.S. Weather Stations, local airports, yacht clubs, Coast Guard, local air pollution control agency, agricultural experiment stations, universities, companies, and individuals.

GO TO STEP 26

26. Was either temperature or amount of precipitation extreme in either direction during the relevant period?

No – GO TO STEP 37
Yes – GO TO STEP 27

27. Are symptoms consistent with symptoms normally associated with the type of environmental stress indicated in step 26?

No – GO TO STEP 37
Yes – GO TO STEP 28

28. Environmental stress is the most likely cause of the observed injury.

STOP

29. There is a chance that a nutrient deficiency is the cause of injury. Send foliar samples to a university or commercial laboratory for testing.

GO TO STEP 30

30. What were results of tests?

Deficient – GO TO STEP 31
Not deficient – GO TO STEP 11

31. The plants were deficient and this deficiency is the most likely cause of the injury.

STOP

32. Are the observed symptoms consistent with symptoms normally associated with the pollutant from the source discovered in step 18?

No – GO TO STEP 84
Yes – GO TO STEP 33

33. The causal pollutant and its likely source are now known. However, in order to confirm the diagnosis do the following:
    a. Conduct a foliar analysis to rule out a nutrient deficiency rather than just rely on the native plants for an answer.
    b. Definitely rule out a disease either by experience or by sending samples to a disease diagnostic laboratory.
    c. Look carefully for an insect pest.
    d. Obtain accurate meteorological data.

    At this point the state extension service should be contacted about the incident.

STOP

34. At this point a definitive answer cannot be determined unless the symptoms of the various pollutants can be accurately discriminated. It is suggested that the first half of the handbook be consulted to assist in the discrimination and that a plant specialist be contacted for additional assistance.

STOP

35. The causal pollutant has just been named. How many of the possible sources emit that pollutant?

One – GO TO STEP 33
More than one – GO TO STEP 36

36. The probable causal pollutant has now been identified, but a single source cannot be named unless the meteorological and topographical data eliminate all but one. It is suggested that an air pollution specialist and a plant specialist be contacted at this point.

STOP

37. Consult the local air pollution control agency to obtain information in the emission inventories concerning the sources of pollutants in the area and the pollutants they can be emitting. Remember that if one of the pollutants under consideration is a photochemical oxidant there will not be a source in the inventory.

GO TO STEP 38

38. Study the local topography of the area, noting terrain features such as mountains, valleys, etc.

GO TO STEP 39

39. Using the information collected on the meteorological conditions, topography, and the emission inventory, is there a possibility that a source(s) in the area emitted a pollutant(s) that could have reached the site of injury?

No – GO TO STEP 84
Yes – GO TO STEP 40

40. A list of possible sources and their possible pollutants now exists. Are the observed symptoms specific for any of these pollutants?

No – GO TO STEP 42
Yes – GO TO STEP 41

41. How many sources are possibly emitting this pollutant?

One – GO TO STEP 33
More than one – GO TO STEP 36

42. Reconstruct the sensitivity chart developed in step 2 using only the possible pollutants based on the meteorological data and information in the emission inventories collected. Is there now any trend?

No – GO TO STEP 45
Yes – GO TO STEP 43

43. How many pollutants remain suspect?

One – GO TO STEP 44
More than one – GO TO STEP 22

44. Are the symptoms consistent with those normally associated with the one remaining pollutant?

No – GO TO STEP 45
Yes – GO TO STEP 46

45. At this point there is no real conclusion you can draw. It is suggested a plant specialist be contacted.

STOP

46. How many sources could possibly be emitting this pollutant?

One – GO TO STEP 33
More than one – GO TO STEP 36

47. The chance exists that a nutrient deficiency is the cause of injury. Send foliar samples to a university or commercial laboratory for testing.

GO TO STEP 48

48. What were results of tests?

Deficient – GO TO STEP 49
Not deficient – GO TO STEP 25

49. The plants are deficient as indicated in the laboratory results. This is probably the cause of the observed symptoms.

STOP

50. Since there is just one species expressing symptoms, there is a good chance that a disease is responsible for the symptoms. Have you had considerable experience in diagnosing plant disease symptomatology?

No – GO TO STEP 54
Yes – GO TO STEP 51

51. Do the symptoms resemble any disease known to you?

No – GO TO STEP 54
Yes – GO TO STEP 52

52. Would this case be considered a classic example of that disease?

No – GO TO STEP 54
Yes – GO TO STEP 53

53. At this point convincing evidence exists that a disease is the cause of the injury.

STOP

54. Send examples of the injured plants to a disease diagnostic laboratory.

GO TO STEP 55

55. At this point other areas should be investigated for additional species with similar symptoms.

GO TO STEP 56

56. Were there any other species with similar symptoms found?

No – GO TO STEP 60
Yes – GO TO STEP 57

57. What were the results of disease tests?

Disease present – GO TO STEP 59
No disease – GO TO STEP 58

58. This choice was anticipated since other affected species have now been located and a disease is not likely to affect more than one species.

GO TO STEP 8

59. Since additional affected species have now been found, the likelihood of a disease is very low. It may be worthwhile to send samples of the other affected species to a laboratory for disease tests as well as to confirm the original test results. If the tests are negative, go to step 8. If the tests still show positive, then a disease must be concluded.

STOP

60. What were the results of disease tests?

Disease present – GO TO STEP 61
No disease – GO TO STEP 62

61. Although there may still be an air pollution problem interacting with the disease identified, the disease must be considered the most likely cause of the injury.

STOP

62. Is the affected species rated sensitive or intermediate to any pollutant or pollutants?

No – GO TO STEP 65
Yes – GO TO STEP 63

63. Are any of the unaffected species in the area rated sensitive or intermediate to that pollutant or pollutants?

No – GO TO STEP 69
Yes – GO TO STEP 64

64. Since these plants are unaffected, then air pollution can be ruled out. If air pollution was the cause, these plants would have been injured also.

STOP

65. Are any unusual events known that could have led to a very high concentration of a pollutant? (e.g., a leak in a passing tank car, etc.)

No – GO TO STEP 85
Yes – GO TO STEP 66

66. Are the observed symptoms consistent with those normally associated with injury caused by that pollutant? (Consult appropriate chapters in this handbook.)

No – GO TO STEP 68
Yes – GO TO STEP 67

67. The causal pollutant and its source have just been identified.

STOP

68. At this point air pollution must be eliminated as a possible cause of the injury on the basis of the following:
    a. Since only one species has been affected, and there is no reason to suspect an accidental fumigation caused the injury, the possibility of an air pollution problem is very remote.
    b. The fact that this species is not rated sensitive to any pollutant indicates a lack of research to back up any evidence of an air pollution episode.

The likely cause can be found perhaps in the realm of nutrient deficiency or environmental stress. Both of these possibilities should be explored, but such exploration is beyond the scope of this book since the intent here is to determine if air pollution is the cause. In this case, there is insufficient evidence to suspect air pollution.

STOP

69. Consult with an air pollution specialist to obtain information in the emission inventories concerning the sources of pollutants in the area and the pollutants they can be emitting. Remember that if one of the pollutants under consideration is a photochemical oxidant there will not be a source in the inventories.

GO TO STEP 70

70. Are there any sources in the area that could possibly be emitting the suspected pollutant or pollutants? Remember that if a photochemical oxidant is under suspicion, the source may be many miles away (refer to Chapter 3).

No – GO TO STEP 75
Yes – GO TO STEP 71

71. Obtain as much data as possible about the following meteorological variables:

a. precipitation
b. temperature
c. wind direction
d. wind speed
e. inversions

Collect data for the following time periods:

a. If the injury appears to be chronic, collect as much as one year's data to determine the prevailing wind.
b. If the injury appears to be acute, collect data from a day or two immediately preceding the onset of symptoms.
c. If a strong possibility exists that the symptoms are environmental stress symptoms, then collect data for the month preceding the onset of symptoms.

Such data are available from the U.S. Weather Stations, local airports, yacht clubs, Coast Guard, local air pollution control agency, agricultural experiment stations, universities, companies, and individuals.

GO TO STEP 72

72. Was either temperature or amount of precipitation extreme in either direction during the relevant period?

No – GO TO STEP 76
Yes – GO TO STEP 73

73. Are the symptoms consistent with symptoms normally associated with the type of environmental stress indicated in step 72?

No – GO TO STEP 76
Yes – GO TO STEP 74

74. Environmental stress is the most likely cause of the injury.

STOP

75. Air pollution can be ruled out since there is no source in the area that could be emitting the suspected pollutant(s).

STOP

76. Study the local topography of the area, noting terrain features such as mountains, valleys, etc.

GO TO STEP 77

77. Using the information you have collected on the meteorological conditions, topography, and the emission inventories, is there a possibility that a source(s) in the area emitted a pollutant(s) that could have reached the site of injury?

No – GO TO STEP 84
Yes – GO TO STEP 78

78. How many sources are under suspicion?

One – GO TO STEP 81
More than one – GO TO STEP 79

79. Do all the possible sources emit the same pollutant?

No – GO TO STEP 82
Yes – GO TO STEP 80

80. Are the observed symptoms consistent with symptoms normally associated with this pollutant? (Consult previous chapters of this book.)

No – GO TO STEP 84
Yes – GO TO STEP 36

81. Are the observed symptoms consistent with symptoms normally associated with the pollutant from that source? (Consult first part of this book.)

No – GO TO STEP 84
Yes – GO TO STEP 33

82. Are the symptoms classical for one of the possible pollutants?

No – GO TO STEP 34
Yes – GO TO STEP 83

83. How many sources could still be emitting the suspected pollutant?

One – GO TO STEP 33
More than one – GO TO STEP 36

84. At this point, air pollution injury is very unlikely and as a result, the cause of the expressed symptoms should be due to one of the other factors mentioned in step 3 (insects, nutrient deficiency, environmental stress, or biotic disease). Since the diagnosis of exactly which of these factors is the cause of the injury is beyond the scope of this book, it is suggested that additional expert assistance be obtained to aid in the diagnosis.

STOP

85. Is the affected species rated tolerant to any pollutant?

No – GO TO STEP 97
Yes – GO TO STEP 86

86. Consult with an air pollution specialist to obtain information in the emission inventories concerning the sources of pollutants in the area and the pollutants they can be emitting. Remember that if one of the pollutants under consideration is a photochemical oxidant there will not be a source in the inventories.

GO TO STEP 87

87. Are there any sources of the suspected pollutant(s) in the area? Remember that if a photochemical oxidant is under suspicion, the source may be many miles away (refer to Chapter 3).

No – GO TO STEP 98
Yes – GO TO STEP 88

88. Obtain as much data as possible about the following meteorological variables:

a. precipitation
b. temperature
c. wind direction
d. wind speed
e. inversions

Collect data for the following time periods:

a. If the injury appears chronic, collect as much as one year's data to determine the prevailing wind.
b. If the injury appears acute, collect data from a day or two immediately preceding the onset of symptoms.
c. If a strong possibility exists that the symptoms are environmental stress symptoms, then collect data for the month preceding the onset of symptoms.

Such data are available from the U.S. Weather Stations, local airports, yacht clubs, Coast Guard, local air pollution control agency, agricultural experiment stations, universities, companies, and individuals.

GO TO STEP 89

89. Was either temperature or amount of precipitation extreme in either direction during the relevant period?

No – GO TO STEP 92
Yes – GO TO STEP 90

90. Are symptoms consistent with symptoms normally associated with the type of environmental stress indicated in step 89?

No – GO TO STEP 92
Yes – GO TO STEP 91

91. Environmental stress is the most likely cause of the observed injury.

STOP

92. Study the local topography, noting terrain features such as mountains, valleys, etc.

GO TO STEP 93

93. Do the meteorological data and the local terrain suggest a buildup of a very high concentration of a pollutant(s) in the area?

No – GO TO STEP 99
Yes – GO TO STEP 94

94. Are the expressed symptoms consistent with symptoms normally caused by one of the suspected pollutants?

No – GO TO STEP 100
Yes – GO TO STEP 95

95. The probable causal pollutant has now been identified.

GO TO STEP 96

96. How many sources are in the right direction and within range to cause the injury?

One – GO TO STEP 33
More than one – GO TO STEP 36

97. At this point air pollution must be eliminated as a possible cause of the injury based on the following:
   a. Since only one species has been affected and there is no reason to suspect an accidental fumigation, the possibility of an air pollution problem is very remote.
   b. The fact that this species is not rated for sensitivity to any pollutant indicates a lack of research to back up any evidence of an air pollution episode.

The likely cause can be found perhaps in the realm of nutrient deficiency or environmental stress. Both of these possibilities should be explored, but such exploration is beyond the scope of this book since the intent here is to determine if air pollution is the cause. In this case there is insufficient evidence to suspect air pollution.

STOP

98. At this point air pollution must be eliminated as a possible cause of the injury for the following reasons:

a. There is only one species affected and this species is not known to be sensitive to any pollutant.

b. This species is tolerant to one or more pollutants, but there is no source of the pollutant(s) in the area.

If air pollution was, in fact, the cause of the injury, there is insufficient evidence to support such a conclusion.

It is suggested that the possibility of a nutrient deficiency or environmental stress condition be explored in order to determine a likely cause. Also, it would be beneficial to solicit additional assistance from a botanist or plant pathologist.

STOP

99. At this point air pollution can be eliminated as a cause since a high concentration would be necessary to cause injury to the affected species. The cause may be found in the realm of nutrient deficiency or environmental stress. Both of these possibilities should be explored, but such exploration is beyond the scope of this book.

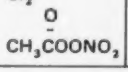
STOP

100. At this point, unless the symptoms can be accurately distinguished as typical of one pollutant, some outside assistance should be sought.

STOP

APPENDIX A
CONVERSION FACTORS

Calculated at Reference Conditions: 25°C., 760 mm Hg

Gas	*Name*	*To convert from ppm to μg/m³ multiply ppm by:*	*To convert from μg/m³ to ppm multiply μg/m³ by:*
NH_3	Ammonia	696	14.40×10^{-4}
HF	Hydrogen fluoride	818	12.20×10^{-4}
CO	Carbon monoxide	1150	8.73×10^{-4}
$CH_2{:}CH_2$	Ethylene	1150	8.72×10^{-4}
NO	Nitric oxide	1230	8.15×10^{-4}
H_2S	Hydrogen sulfide	1390	7.18×10^{-4}
HCl	Hydrogen chloride	1490	6.71×10^{-4}
F_2	Fluorine	1550	6.44×10^{-4}
NO_2	Nitrogen dioxide	1880	5.32×10^{-4}
O_3	Ozone	1960	5.10×10^{-4}
SO_2	Sulfur dioxide	2620	3.82×10^{-4}
Cl_2	Chlorine	2900	3.45×10^{-4}
 $CH_3\overset{\overset{O}{\|}}{C}OONO_2$	PAN	4947	2.02×10^{-4}

NOTE: All factors rounded to three significant figures.

To convert other gases to ppm (μℓ/ℓ) or μg/m³, one of the following equations may be used:

$$\text{ppm to } \mu g/m^3\text{: } \mu g/m^3 = \frac{\text{ppm} \times \text{MW} \times 10^3}{\text{MV}}$$

$$\mu g/m^3 \text{ to ppm: ppm} = \frac{\mu g/m^3 \times \text{MV} \times 10^{-3}}{\text{MW}}$$

MW = Molecular Weight

MV = Molar Volume (24.46 ℓ/mole at 25°C., 760 mm Hg)

APPENDIX B
NATIONAL PRIMARY AND SECONDARY AIR QUALITY STANDARDS

Primary standards were set at a level which would prevent health effects from occurring. They are based on the best information available at the time of their establishment. Secondary standards were set at a level which would prevent deleterious effects from occurring on vegetation and materials such as fabrics, paints, masonry, etc. These standards were also established on the basis of best available information at the time. It should be noted, however, that the standards, both primary and secondary, were established based on data which took into consideration the presence of only one gas in the exposure atmosphere. This is seldom the case in nature. Receptors may be exposed to a number of pollutants of various concentrations, simultaneously, sequentially, or intermittently. It is possible that, as more information becomes available, these standards may need adjustment.

It should also be noted that these are federal standards, and some states and municipalities may have more stringent standards. The reader is cautioned to inquire about state or local regulations.

National Primary and Secondary Air Quality Standards*

Pollutant	*Duration*	*Primary Standard*	*Secondary Standard*
Sulfur Dioxide	Annual Arithmetic Mean	80 $\mu g/m^3$ (0.03 ppm)	---
	Max. 24 hour	365 $\mu g/m^3$ (0.14 ppm)	---
	Max. 3 hour	---	1300 $\mu g/m^3$ (0.5 ppm)
Photochemical Oxidants	Max. 1 hour	160 $\mu g/m^3$ (0.08 ppm)	Same
Nitrogen Dioxide	Annual Arithmetic Mean	100 $\mu g/m^3$ (0.05 ppm)	Same
Hydrocarbons	Max. 3 hour concentration (6-9 AM) not to exceed once per year	160 $\mu g/m^3$ (0.24 ppm)	Same
Particulate Matter	Annual Geometric Mean	75 $\mu g/m^3$	60 $\mu g/m^3$
	Max. 24 hour concentration not to exceed once per year	260 $\mu g/m^3$	150 $\mu g/m^3$

Pollutant	*Duration*	*Primary Standard*	*Secondary Standard*
Carbon Monoxide	Max. 8 hour concentration not to exceed once per year	10 mg/m^3 (9 ppm)	Same
	Max. 1 hour concentration not to exceed once per year	40 mg/m^3 (35 ppm)	Same

*Based on Federal Register, Vol. 36, No. 84, April 30, 1971, pp. 8186-8201. (Secondary standards for Sulfur Dioxide revised by Federal Register, Vol. 38, No. 178, September 14, 1973)

APPENDIX C

COMPOSITE SENSITIVITY CHART AND SCIENTIFIC NAMES OF PLANTS

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Acacia *Acacia sp.*												T
Adonis *Adonis sp.*									T			
Ageratum *Ageratum sp.*												I
Alder *Alnus sp.*	I		S									
European Black *A. glutinosa (L) Gaertn.*									I			
Alfalfa *Medicago sativa L.*	S	I	S	T	S		S					
Aloe *Aloe sp.*										T		
Anthurium *Anthurium sp.*		T										
Apple *Malus sylvestris Mill.*		T	S	I	S	S						
Apricot *Prunus armeniaca L.*	T											
Chinese *P. armeniaca L. var. Chinese*	I			S								
Moorpark *P. armeniaca L. var. Moorpark*				I								
Royal *P. armeniaca L. var. Royal*				S								
Tilton *P. armeniaca L. var. Tilton*				I								

S = Sensitive; I = Intermediate; T = Tolerant

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Arborvitae *Thuja occidentalis L.*	T	T		T					T			
Ash *Fraxinus sp.*						T						
American Mt. *Sorbus americana L.*		T										
European Mt. *S. aucuparia L.*	S			I								
Green *Fraxinus pennsylvanica Marsh.*	S	T		I								
Modesto *F. velutina Torr.*				T								
White *F. americana L.*	S	T										
Asparagus *Asparagus officinalis L.*				T	T							
Aspen, Large-Toothed *Populus, grandidentata Michx.*			S									
Quaking *P. tremuloides Michx.*	S		S	I								
Aster *Aster sp.*		S	S	I		I						
China *Callistephus chinensis*												T
Avocado *Persea americana Mill.*	T											
Azalea *Rhododendron sp.*		T		I	S		T			I		
Campfire *R. kaempheri "campfire" (Planch) Wils. Hort.*	S											
Chinese *R. molle sp.*	T											

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Hino *R. hinodegiri Hort.*	S											
Korean *R. poukhanensis (Nakai.) Gable*	S											
Snow *R. mucronatum*	S											
Bachelor's Button *Centaurea cyanus L.*			S									
Barley *Hordeum vulgare L.*	S	I	S		S							
Young				S								
Mature				I								
Basil, Sweet *Ocimum*		S										
Basswood *Tilia americana L.*		T								I		
Bean *Phaseolus sp.*	S	S	S							S		
Bush *P. vulgaris L.*					I	T						
Field *Fragaria chiloensis*					I							
Lima *Phaseolus limensis Macf.*		T										
Pinto *P. vulgaris L.*					S		I		I			
Scotia *P. vulgaris L.*							I					
Snap *P. vulgaris L.*				T								

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Beech *Fagus sp.*												
European *F. sylvatica L.*	T											
Purple *F. sylvatica atropurpurea*					T							
Beet *Beta sp.*	T		S									
Sugar *B. vulgaris L.*		I										
Table *B. vulgaris L.*		I										
Begonia *Begonia sp.*	I	T					T			I		
Tuberous *Begonia sp.*					S							
Birch *Betula sp.*						S						
Cutleaf *B. pendula var. gracilis Roth*				T								
European White *B. pendula Roth*	T	T			S							
White *B. papyrifera Marsh*			S	T								
Blackberry *Rubus sp.*							S					
Blueberry *Vaccinium sp.*				S								
Bougainvillea *Bougainvillea spectabilis*					S							
Box, Japanese *Buxus sp.*	T											

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Boxelder *Acer negundo L.*	I			S		S	S					
Brazilian Pepper Bush *Shinus terebinthifolius*			S									
Bridalwreath *Spiraea prunifolia Sieb. & Zucc.*	S			T								
Brittlewood *Melaleuca leucadendra*					I							
Broccoli *Brassica oleracea L.*	I	T	S									
Bromeliad *Bromeliad sp.*		T										
Browallia *Browallia sp.*	S											
Burdock *Arctium sp.*				T								
Butterflyweed *Asclepias tuberosa L.*										S		
Cabbage *Brassica oleracea L.*	I	T	T	T	T	T						
Cacti *Cactaceae sp.*		T					T					
Calendula *Calendula sp.*		T										T
Camellia *Camellia sp.*		T		T						I		
Cape Jasmine *Gardenia radicans*					I							
Careless Weed *Amaranthus palmeri S. Wats.*			S									
Carissa *Carissa carandas*					T							

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Carnation *Dianthus caryophyllus sp.*	I	T										I
Carrot *Daucus carota L.*	I	I	S	T	S							
Catalpa *Catalpa bignonioides Walt.*	I											
Cauliflower *Brassica oleracea botrytis L.*		T		T								
Cedar *Cedrus sp.*			T			I						
Incense *Libocedrus decurrans Torr.*	I											
Celery *Apium graveolens L.*		S	T	T	I							
Cheeseweed *Malva parviflora L.*		I			I		I	S			T	
Chenopodium *Chenopodium sp.*							T					
Cherry *Prunus sp.*						I				T		
Bing *P. avium L. var. Bing*	S											
Black *P. serotina Ehrh.*									I			
Choke *P. virginiana L.*				I		I						
Flowering *P. serrulata Lindl.*				I								
Ground *P. tructiosa L.*		S										
Lambert *P. avium L. var. Lambert*	I											
Sweet *P. avium L.*				I								

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Chickweed *Stellaria media*		S	S	S	I		S	I			T	
Chrysanthemum *Chrysanthemum sp.*	I	T		T			T		I			
Cinquefoil *Potentilla sp.*										S		
Citrus *Citrus sp.*	S		T									
Clover *Trifolium sp.*		S										
Crimson *T. incarnatum L.*				I								
Red *T. pratense L.*	S		S		S							
Spring *T. incarnatum*					S							
Sweet *Melilotus sp.*				I								
Yellow *Melilotus officinalis Lam.*			S									
Coleus *Coleus sp.*	S	T						T				
Columbine *Aquilegia sp.*									S	I		
Corn *Zea mays L.*		T	T		I	I	I					
Field *Z. mays L.*	I			SI			T					
Sweet *Z. mays L.*	S			SI								
Cornflowers *Centaurea cyanus L.*									S			

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Cosmos *Cosmos sp.*			S							I		
Cotoneaster *Cotoneaster sp.*										I		
Rock *C. horizontalis Decne.*	S											
Spreading *C. divaricata Rehd. & Wils.*	S											
Cotton *Gossypium sp.*	T	T	S	T								I
Cowpea *Vigna sinensis Savi*	I						T					I
Crabapple *Malus baccata Borkh.*	I						S					
Croton *Codiaeum variegatum*					T					T		
Cucumber *Cucumis sativus L.*	I	T	S	T			I					
Currant *Ribes sp.*				T								
Cyclamen *Cyclamen sp.*		T										
Dahlia *Dahlia sp.*		S		I	I		I					T
Daisy *Chrysanthemum leucanthemum*					T				S			
Dandelion *Taraxacum officinale Web.*					I		I	I			T	
Descurainia *Descurainia californica (Gray) Shultz*	T											

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Dock *Rumex sp.*			S	T								
Sour *R. crispus L.*		I										
Dogwood *Cornus sp.*		T		T		S						
Gray *C. reacemosa Lam.*	T											
White *C. florida L.*	T											
Eggplant *Solanum melongena L. var. esculentum Nees.*				T		T						
Elder *Robinia pseudoacacia*					T							
Elderberry *Sambucus sp.*				T		S						
Elm *Ulmus sp.*					T							
American *U. americana L.*				T								
Chinese, Siberian *U. pumila L.*				T								
Endive *Cichorium endivia L.*	I	S										
Euonymus, Dwarf Winged *Euonymus sp.*	T											
Fern												
Boston *Nephrolepis exaltata Schott.*										S		

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Bracken *Pteridium aquilinum L.*			S									
Holly *Cyrtomium falcatum Smith*											S	
Fir *Abies sp.*												
Big-Cone Douglas *Pseudotsuga menziesii (Mirb.) Franes*	I											
Balsam *A. balsamea Mill.*	T	T			I		T		T			
Douglas *Pseudotsuga taxifolia Br. H.*	T	T		S					T			
Grand *A. grandis (Dougl.) Lindl.*				I								
White *A. concolor Hoopes.*	IT	T										
Firethorne *Pyracantha coccinea Depp.*				T								
Laland's *P. coccinea laland Depp.*	T											
Forget-me-not *Myosotis sp.*												T
Forsythia *Forsythia sp.*						S				I		
Lynwood Gold *F. intermedia spectabilis "Lynwood Gold" Koehn*	I											
Four-o-clock *Mirabilis jalapa L.*			S									
Fuchsia *Fuchsia sp.*	T	S			I					I		

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Gardenia *Gardenia jasminoides*					I					I		
Geranium *Geranium sp.*	T			I			T			I		
Gingko *Gingko biloba*					T							
Gladiolus *Gladiolus sp.*	T		S	S	T	I						
Gloxinia *Sinningia sp.*	T											
Goldenrod *Solidago sp.*				I					T			
Gomphrena *Gomphrena globosa L.*							S					
Goosefoot, Nettle-Leaf *Chenopodium sp.*				I	T		I	I			S	
Grape *Vitis sp.*	S					S						
Concord *V. labruscana Bailey*	S			I								
European *V. vinifera L.*				S								
Oregon *Mahonia repens (Lindl.) Don*				S								
Thompson Seedless *Vitis sp.*	I											
Grapefruit *Citrus paradisi Macf.*				I								
Grass												
Annual Blue *Poa annua L.*		S	S		I		I	S			T	
Bent *Agrostis palustris Huds.*	S											

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Brome *Bromus inermis Leyss.*	S											
Crab *Digitaria ischaemum Schreb.*	S			S								
Johnson *Sorghum halepense Pers.*				I								
Kentucky Blue *Poa pratensis*					T		T	I			T	
Orchard *Dactylis glomerata L.*	S											
Gum, Black *Nyssa sylvatica (Marsh.) Pepp.*	T											
Hazel *Corylus sp.*			S									
Heath *Erica sp.*					T							
Hemlock *Tsuga sp.*						I						
Eastern *T. canadensis Carr.*	T	T										
Hibiscus *Hibiscus sp.*					S							
Hickory *Carya sp.*						S						
Holly *Ilex sp.*												
American *I. opaca Ait.*										I		
American (Male)	T											
American (Female)	T											

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Chinese *I. chinensis Loes.*										T		
Hetz's Japanese *I. crenata Hetzi (Thurb.) Hetz.*	T											
Honeysuckle, Blue-leaf *Lonicera zabeli Rehd.*	I											
Hornbeam, European *Tilia sp.*					T							
Horse Chestnut *Aesculus hippocastanum L.*							S					
Hydrangea *Hydrangea sp.*										S		
Hypericum *Hypericum sp.*	I											
Impatiens *Impatiens sp.*	T											
Iris *Iris sp.*				S					T			
Ivy *Hedera sp.*		T								T		
English *H. helix L.*	T											
Ixora *Ixora coccinea*					I							
Jerusalem Cherry *Solanum pseudo-capsicum L.*	T			SI								
Jimsonweed *Datura stramonium L.*		S										
Juniper *Juniperus sp.*				T								
Shore *J. conferta*					T							

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Western *J. occidentalis Hook.*	T											
Kohlrabi *Brassica oleracea cv. gongylodes*			S		T							
Lamb's Quarter *Chenopodium album L.*	T	I		SI	T	S	T	S			S	
Larch *Larix sp.*			S									
European *L. decidua Mill.*	I	T			S							
Japanese *L. leptolepsis (Sieb. & Zucc.) Gord.*	I	T			S							
Western *L. occidentalis Nutt.*				S								
Larkspur *Delphinium sp.*									T			I
Laurel, Mountain *Kalmia latifolia L.*	T			T								
Leek *Allium porrum*					S							
Lemon *Citrus lemon Burm.*	T			I								
Lettuce *Lactuca sativa*	T	S	S		S							
Prickly *L. scariola L.*			S									
Ligustrum *Ligustrum lucidum*					I							
Lilac *Syringa vulgaris L.*			T	I								

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Chinese *S. chinensis L.*	S											
Common *S. vulgaris L.*	S	T										
Lily *Lilium sp.*		T										I
Calla *Zantedeschia aethiopica Spreng.*										I		
Easter *Lilium eximum Baker*										I		
Of-the-Valley *Convallaria majalis*					T				T			
Plantain *Hosta plantaginea (Lam.) Archers*					T				T			
Linden *Tilia sp.*			T		I	S						
American *T. americana L.*	T			T								
Little-Leaf *T. cordata Mill.*	T			I								
Lobelia *Lobelia sp.*												T
Locust												
Black *Robinia pseudoacacia*	T			T	T							
Honey *Gleditsia triacanthos L.*	S	T										
London Plane Tree *Platanus acerifolia (Ait.) Willd.*				T		S						
Lupin *Lupinus sp.*									T			

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Maple *Acer sp.*			T									
Hedge *A. campestre L.*				I								
Japanese *A. palmatum Thumb.*					I					I		
Norway *A. platanoides L.*	T	T		I	I	S			I			
Red *A. rubrum L.*	T											
Silver or White *A. saccharinum L.*		T		I								
Sugar or Rock *A. saccharum Marsh.*	T	T					S		I			
Marigold *Tagetes sp.*	T											
African *T. erecta L.*												S
Mignonette *Reseda lutea L.*							T					
Milo Maize *Sorghum vulgare Pers.*				SI								
Mimosa *Albizzia julibrissin Duratz.*	T									S		
Mimulus *Mimulus sp.*		S										
Mint *Mentha sp.*	T	S										
Mulberry *Morus sp.*						I						
Red *M. rubra L.*				I								
Muskmelon *Cucumis melo L.*	S		T									

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Mustard *Brassica sp.*		S			S		S	S			T	
Cultivated			S									
Wild			S									
Myrtle *Myrtus sp.*							T					
Narcissus *Narcissus sp.*		T		I								
Nasturtium *Tropaeolum sp.*							I					
Nettle, Little-Leaf *Urtica urens L.*		S										
Night Shade *Solanum nigrum L.*			S	T								
Oak *Quercus sp.*				T	T					I		
Black *Q. velutina Lam.*	I					S						
Bur *Q. macrocarpa Michx.*	T											
English *Q. robur L.*	T	T										
Gambel *Q. Gambelii Nutt.*	S											
Northern Red *Q. rubra L.*	T	T										
Pin *Q. palustris Muench.*	I	T				I	S					

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Red *Q. rubra L.*						I			T			
Scarlet *Q. coccinea Muench.*	I											
Shingle *Q. imbricaria Michx.*	T											
White *Q. alba*	S	T										
Oats *Aven sativa L.*	S	S	S		ST							
Mature				I								
Wild		S										
Young				I								
Oleander *Nerium oleander*					S							
Olive, Russian *Elaeagnus angustifolia L.*				T								
Onion *Allium cepa sp.*	S	T	T		T		I					
Orange *Citrus sinensis Osbeck*				I	I							
Mock *Philadelphus sp.*				T								
Mock Sweet *P. coronarius L.*	I											
Orchid *Orchidaceae sp.*		T										S
Oriental Poppy *Papaver orientale*									S			
Oxalis *Oxalis sp.*							T			S		

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Pachysandra *Pachysandra terminalis Sieb. & Zucc.*	T											
Pagoda, Japanese *Sophora japonica L.*	T											
Parsley *Petroselinum crispum NYM*	I				S							
Parsnip *Pastinaca sativa L.*	I											
Peach *Prunus persica Batsch.*	T					I				I		
Fruit				S								
Foliage				I								
Peanut *Arachis hypogaca*	I											
Pear *Pyrus communis L.*				T	S	T						
Bartlett *P. communis L. var. Bartlett*	T											
Pea *Pisum sativum L.*	I		S	T	S							
Peony *Paeonia sp.*				I		T						
Pepper *Piper nigrum L.*	I	S					T					I
Bell *Capsicum frutesceans L.*				T								
Periwinkle *Vinca minor L.*	T	T										
Persimmon *Diospyros virginiana L.*										I		
Petunia *Petunia sp.*	S	S		T	I		I					

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Pheasant's Eye Pink *Dianthus sp.*									T			
Phlox *Phlox sp.*												
Caespitose									S			
Paniculata									T			
Piggy-Back Plant *Tolmiea menziesii Torr. & Gray*	T											
Pigweed *Amaranthus retroflexus L.*		S		I	T		T	S			T	
Pieris, Japanese *Pieris japonica*	T											
Pine *Pinus sp.*					T		T					
Austrian *P. nigra Arnold*	S	T	S						T			
Coulter *P. coulteri D. Don*	S											
Digger *P. sabiniana Dougl.*	T											
Eastern White *P. strobus L.*	S	T										
Jack *P. banksiana Lamb.*	S		S									
Jeffrey *P. jeffreyi A. Murr.*	S											
Knobcone *P. attenuata Lemm.*	I											
Lodgepole *P. contorta Loud.*	I			T								
Monterey *P. radiata D. Don*	S											

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Mugho *P. mugho Turra.*				T								
Pitch *P. rigida Mill.*	I	T										
Ponderosa *P. ponderosa Laws.*	S			S								
Red *P. resinosa Ait.*	T	T	S									
Scotch *P. sylvestris L.*	I	T		S								
Single-Leaf Pinyon *P. monophylla Torr. & Frem.*	T											
Sugar *P. lambertiana Dougl.*	I											
Torrey *P. torreyana Perry*	I											
Virginia *P. virginiana Mill.*	S	T										
White *P. strobus L.*			S						I	I		
Pittosporum *Pittosporum tobira*					I							
Plantain *Musa paradisiaca L.*				T								
Plum *Prunus sp.*												
Bradshaw *P. domestica L.*				S								
Flowering *P. cerasifera Ehrh.*				IT								
Poinsettia *Euphorbia pulcherrima*	I											
Polygonum *Polygonum sp.*							T					

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Poplar *Populus sp.*												
Balsam *P. balsamifera L.*				T								
Carolina *P. canadensis Muench.*				I								
Hybrid *P. sp.*	S	T										
Lombardy *P. nigra var. italica Muench.*				I								
Tulip *Liriodendron tulipifera L.*	S	T							S			
Potato *Solanum tuberosum L.*	S		T	T	I	I						
Sweet *Ipomoea batatas (L) Lam.*	T		S	SI								
Primrose *Primula sp.*		S										
Privet *Ligustrum sp.*			T	T		I				SI		
Amur North *L. amurense*	T											
Lodense *L. vulgare var. pyramidale*	S											
Common *L. vulgare*	I											
Prune Tree, Italian *Prunus domestica L.*				S								
Purslane *Portulaca oleracea L.*				T								
Pyracantha *Pyracantha sp.*					S							

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Radish *Raphanus sativus L.*	S	T	S				S					
Ragweed *Ambrosia artemisiifolia L.*			S			I						
Ranunculus *Ranunculus sp.*		S										
Raspberry, Red *Rubus idaeus L.*				T								
Redbud, Eastern *Cercis canadensis L.*	I											
Redwood *Sequoia sempervirens Endl.*	T											
Rhododendron *Rhododendron sp.*				I		I						
Carolina *R. carolinianum*	T											
Catawba *R. catawbiense*					I							
Catawbiense Album *R. catawbiense album (Michx.) Hort.*	I											
Nova Zembla *R. nova zembla Hort.*	I											
Roseum Elegans *R. roseum elegans Hort.*	I											
Rhubarb *Rheum rhaponticum L.*		T	S		S	T						
Rice *Lactuca longifolia Lam.*	T											
Rose *Rosa odorata Sweet.*				I	S	I	S					I
Rye *Secale cereale L.*	S				I							
Mature				I								

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Young				I								
Safflower *Carthamus tinctorius L.*	S											
Salvia *Salvia sp.*										I		
Sarcococca *Sarcococca sp.*										T		
Saxifrage *Saxifraga sp.*										I		
Sequoia, Giant *Sequoiadendron gigantea Buchholz*	T											
Serviceberry *Amelanchier alnifolia*				I								
Silverberry *Eleagnus argentea Pursh. Nutt.*	I											
Smartweed *Polygonum sp.*	S			SI								
Snapdragon *Antirrhinum majus L.*	T	S			S							I
Snowberry *Symphoricarpos sp.*	S											
Sorghum *Sorghum vulgare Pers.*	I	T				T						
Sorrel *Rumex sp.*						S						
Sow Thistle *Sonchus sp.*			S									
Soybean *Glycine max. Merr.*	S	I		IT								
Spinach *Spinacea oleracea L.*	S	I	S	T								
Spirea *Spiraea sp.*	T											

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Spruce *Picea sp.*					I	I						
Black Hills *P. glauca densata Bailey*	T	T										
Blue *P. pungens Engelm.*		T		SI								
Colorado Blue *P. pungens Engelm.*	T											
Norway *P. abies Karst.*	T	T							T			
White *P. glauca Voss.*	T	T		I								
Squash *Curcurbita pepo L.*		T	S				I					
Summer *C. pepo L.*				T								
St. John's Wort *Hypericum perforatum L.*				S								
Stevia *Piqueria trinervia Cav.*	I											
Strawberry *Fragaria sp.*	T	T		I						I		
Sugar Cane *Saccharum officinarum L.*				T								
Sumac *Rhus sp.*	S					S						
Smooth *R. glabra L.*				I								
Staghorn *R. typhina L.*				I								
Sunflower *Helianthus sp.*				I	S		S	S		S	T	
Sweet Pea *Lathyrus odoratus*			S		S							T

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Sweet William *Dianthus barbatus L.*			S	I					T			
Sweetgum *Liquidambar styraciflua L.*	I	T		T		I	S					
Swiss Chard *Beta vulgaris var. cicla L.*		S	S									
Sycamore *Platanus sp.*				T								
American *P. occidentalis L.*	S											
Tangerine *Citrus reticulata Blanco.*				I								
Timothy *Phleum pratense L.*	I											
Tobacco *Nicotiana tabacum L.*	S	I		T	S	S	S			I		
Tomato *Lycopersicum esculentum Mill.*	S	S	S	I	I	S	I	T	S	I		I
Tree-of-Heaven *Ailanthus altissima Swingle*	S			T		S	S					
Tulip *Tulipa gesneriana L.*			S	S			S					
Tulip Tree *Liriodendron tulipifera L.*	S	T							S			
Turnip *Brassica rapa L.*	I		S									
Vetch, Spring *Vicia sativa*					S							
Viburnum *Viburnum sp.*	T									I		

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Eth-ylene
Korean Spice *V. carlesii*	T											
Linden *V. dilatatum Thune*	I											
Tea *V. setigerum Hance.*	I											
Vinca *Vinca sp.*										I		
Violet *Viola sp.*			S	I								T
Virginia Creeper *Parthenocissus quinquefolia Planch.*	T			T			S					
Walnut *Juglans sp.*												
Black *J. nigra L.*	T			I								
English *J. regia L.*	SI			I								
Wandering Jew *Zebrina sp.*							S					
Wheat *Triticum aestivum L.*	S	I	S	I	I							
Buck *Fagopyrum esculentum Muench.*	S		S				I	T				
Willow *Salix sp.*			S	T						S		
Weeping *S. babylonica L.*	I											
Wisteria *Wisteria sp.*						S						

APPENDIX C (Continued)

PLANT	O_3	PAN	SO_2	F	NO_2	2,4-D	Cl	NH_3	HCl	Hg	H_2S	Ethylene
Yellow Wood *Cladrastis lutea Koch.*						S						
Yew *Taxus baccata*					T	I						
Dense *T. densiformis Hort.*	T											
Hatfield's Pyramidical *T. media hatfieldi Rehd.*	T											
Spreading Japanese *T. cuspidata Sieb. & Zucc.*				I								
Zinnia *Zinnia sp.*	T		S			S	S					I

APPENDIX D

BIO-INDICATORS

Certain plant species, or more specifically, certain cultivars or clones have been used as sensitive indicators of air pollutants. Their use has been particularly useful for "monitoring" in remote areas where power for instruments is not available and for determining the environmental conditions which are conducive to plant injury. Their major limitations are lack of specificity and lack of identifiable growth and reproductive effects. They have proven useful, for example, in delineating extent and frequency of the Los Angeles photochemical smog. In the latter study, annual bluegrass and pinto bean were used. Tobacco has been used by many, and has served as an excellent index of oxidants. Lichens have been used widely in Germany and Canada.

Examples of bio-indicators used to detect ozone include Bel W-3 tobacco (Plate 3-2), certain varieties of pinto beans, white cascade petunia, white ash (Plate 3-18), and certain clonal lines of eastern white pine (Plate 3-23).

A number of researchers have reported that the centers of many cities and certain industrial areas are free, or nearly so, of epiphytic lichens and mosses. The usual situation is to find these epiphytes increasing in numbers and species, as well as in vigor, as the distance from the city center or the source of the air pollution increases. These city "deserts" are believed to be due to SO_2 phytotoxicity, although reduced sunlight and other factors cannot, at present, be ruled out as causes.

In recent years, procedures have been developed for mapping Index of Atmospheric Purity (IAP) lines around city centers or point sources of SO_2. Data for IAP lines are derived from study of the number of lichen species in the area and examination of individual species at selected stations. For such a study, a tree species is selected, based on its frequency and distribution in the area. The trees should be large (0.5 to 1 meter in diameter), not shaded by other objects, and not located in an area where a harmful local atmospheric factor (other than the one to be studied) might exert an influence. Usually, data are taken from 10 to 15 such trees at each station. The lichen data should be taken over the same areas of the tree trunks at all stations (ground to 2-1/2 meters).

Each lichen species is assigned a "frequency-coverage number" *(f)* at each station. This number is usually between 1 and 5, and is assigned as follows:

1. Rare occurrence at the site area and a low degree of coverage.
2. Very infrequent occurrence and a low degree of coverage.
3. Infrequent occurrence or a medium degree of coverage.
4. Frequent occurrence or a high degree of coverage on some trees.
5. Very frequent occurrence and a high degree of coverage on most trees.

Each species is also assigned in "ecological index number," *q*. At each station where the species in question occurs, the number of other epiphyte species present is determined. The number of other species present is then' totalled for all stations, and divided by the number of stations, to find the *average* number of other species present. This average is *q*.

q for a species = average number of other epiphyte species
occurring at stations where species
under study is found

For example, a species of lichen in one study occurred at 245 of 349 stations. The number of companion species at these 245 stations ranged from 4 to 32, and averaged 15. Thus, $q = 15$; $q \times f$ (ecological index x frequency-coverage) is divided by 10 to give a convenient, manageable number. The IAP for each station is one-tenth the sum of the $q \times f$ values for each species present at that station. The IAP for all stations may then be plotted on a map, and isotoxic zones can be defined by linking stations having indexes within a certain range.

Since the effect of SO_2 seems to be to kill or retard growth of these epiphytes, their presence and well-being provides a measure of the total physiological effect of the pollutant on the epiphytes studied. Table D-1 records lichen species, their occurrence or non-occurrence in each of five IAP zones around large point-sources of SO_2, and the q or tolerance values of the lichens studied. High q values mean the lichen is sensitive to SO_2.

Table D-1
Distribution of Epiphytic Lichens in Various IAP Zones*

		ZONES				
	q	I	II	III	IV	V
Alectoria americana	16				x	
A. nidulifera	22				x	x
Bacidia chlorococca	10	x	x	x	x	x
Buellia stillingiana	27					x
Caloplaca cerina	19				x	x
Candelaria concolor	21					x
Candelariella vitellina	19		x	x	x	x
Cetraria ciliaris	27					x
C. pinastri	17		x	x	x	x
C. saepincola	14		x	x	x	x
Cladonia coniocraea	12		x	x	x	x
C. cristatella	11			x		
C. fimbriata						x
Evernia mesomorpha	18		x	x	x	x
Hypogymnia physodes	16		x	x	x	x
Lecanora expallens	13		x	x		x
L. saligna	12	x	x	x	x	x
L. subfusca (coll.)	20				x	x
L. subfusca variolosa	16		x	x	x	x
L. subintricata	18		x		x	x
L. symmicta	21				x	x
Lecidea nylanderi	22					x
Lepraria aeruginosa	11	x		x		x
Parmelia exasperatula	18		x	x	x	x
P. olivacea	21			x		x

	q	ZONES I	II	III	IV	V
P. rudecta	19					x
P. septentrionalis	24				x	x
P. sulcata	13	x	x	x	x	x
P. trabeculata	28					x
Parmeliopsis ambigua	14		x	x		x
Pertusaria multipunctata	14			x		
Physcia aipolia	24				x	x
P. adscendens	20			x		x
P. farrea	24					x
P. grisea	20			x	x	x
P. orbicularis	17					x
P. stellaris	19			x	x	x
Ramalina fastigiata	28					x
Rinodina hallei	20			x	x	x
R. papillata	27					x
Usnea sp.	21				x	x
Xanthoria fallax	17					x
X. polycarpa	19					x
TOTAL		4	14	21	23	40

*From LeBlanc, F. The epiphytic vegetation of *Populus balsamifera* and its significance as an air pollution indicator in Sudbury, Ontario. *Canadian Journal of Botany*, 50:519-528.

Bio-indicator plants may be planted (or transplanted in the case of lichens) in polluted areas and observed periodically for specific symptom types. Such observations may reveal the presence, and perhaps even the relative importance of various pollutants in a given area. If indicator plants are established in "air pollution gardens" scattered over a wide geographic area, the distribution of various pollutants may be determined. Such gardens are also useful youth projects and make good demonstration plots for showing examples of air pollution injury to plants. Table D-2 shows some of the more frequently used bio-indicators.

Certain types of plants may also be used to indicate the amount of a pollutant which has been taken in or absorbed by the plant – even though the plant may not show injury. This type of sampling followed by chemical analysis has been used extensively to determine patterns of exposure or dispersion of fluorides.

The concentration of fluoride in vegetation can be mapped in order to compare one site with another or to determine fluoride dispersion over a large area. The considerations mentioned above concerning superficial fluoride should be applied. Age, location, and portion of tissue sampled can also affect the result. Thus, a uniform method for the selection and preparation of foliage must

Table D-2

List of Common Bio-indicators and Pollutant(s)
to Which They Are Sensitive*

Bio-indicator	*Pollutant*
Alfalfa	SO_2
Gladiolus*	HF, F
Petunia, white cascade	Oxid., PAN
Pinto bean	O_3, PAN
Tobacco, Bel W-3	O_3
White pine*	O_3, SO_2
White ash	O_3
Lichens	SO_2
Bluegrass	Oxid.
Aspen	SO_2
Bracken fern	SO_2

*Certain cultivars or clones only.

be used. The same species should be sampled in each area because different species accumulate atmospheric fluoride at different rates. If one species does not exist at all stations and two or more species must be used, samples should be taken from all species at sites where they overlap to afford a comparison between them. The sites from which samples are obtained should be chosen systematically with respect to the geometry of the map or the expected or predicted pattern of dispersion. More than one sample should be collected at each site in both the affected and remote areas during the first visit. Once the site for sampling has been reached, the cost of additional samples is relatively small, and all samples need not be analyzed. But should variability or some analytical problem arise, it is easier and cheaper to obtain another sample from those stored on the shelf than to resample in the field.

Evaluation of the Fluoride Concentration of Forage

The most important objective often is to judge whether or not the forage in a pasture has accumulated enough fluoride to constitute a hazard to livestock or to violate a legal standard. The methods used for sampling will be determined by two sets of criteria: biological and statistical. Biological criteria are more important with respect to obtaining samples that are representative of the vegetation. If the field contains a mixture of species, the sample should reflect the relative amounts of the species present because different species accumulate fluoride at different rates. The same kind of consideration is also appropriate if the crop contains plants of different ages. The species eaten should also be considered; since thistle and certain other species are rarely eaten, sampling them gives an erroneous value. Unless care is taken in cutting the sample, either at the height mown or cropped, the inclusion or exclusion of older foliage may bias the results. Unless specified by a standard, the sample should not be washed because particulate or superficial fluoride is available for ingestion by livestock. However, care should be taken that the presence of road dust or soil in the sample does not bias the results.

Whenever possible, forage should be sampled just prior to harvest, but if it cannot be, owing to the schedule and plan of sampling, the following factors might be considered in relation to an evaluation of the result. Precipitation can lower the fluoride content. A period of slow growth, due to climate or maturity of the crop, can increase the concentration because of the lack of new foliage, which is lower in fluoride. Similarly, rapid growth may lower the concentration through growth dilution or through senescence and loss of older fluoride-containing leaves.

The actual plan of sampling and number of samples taken is usually not known before the first sampling time. However, the initial samples should be taken in such a way that information can be derived that will allow the most efficient schedule and pattern of sampling to be employed for subsequent sampling periods.

Selected Reference

Heck, W. W. The use of plants as indicators of air pollution. *International Journal of Air and Water Pollution,* 10:99-111, 1966.

List of Commonly Used Bio-indicators

For ozone
- Petunia (certain cultivars only)
- Pinto bean
- Tobacco (Bel W-3)
- White pine (certain strains only)

For PAN
- Pinto bean
- Petunia (some white-flowered cultivars)

For Fluorides
- Gladiolus (c.v. Elizabeth the Queen)
- Iris
- Ponderosa pine

For SO_2
- Alfalfa
- White pine (certain strains only)
- Bean
- White birch

APPENDIX E

GLOSSARY OF AIR POLLUTION AND BOTANICAL TERMS

Abaxial	facing away from the axis of stem; usually refers to the undersurface of a leaf blade.
Abscission	dropping of leaves or other plant parts.
Acute injury	injury, usually involving necrosis, which develops within several hours to a few days after a short-term exposure to a pollutant, and expressed as fleck, scorch, bifacial necrosis, etc.
Adaxial	facing toward the axis of stem; usually refers to the upper surface of a leaf blade.
Aerosol	particles or liquid droplets usually smaller than 100 μ in diameter suspended in air.
Ambient	prevailing condition of the atmosphere at a given time, the outside air.
Area source	geographical location from which pollutants originate and are transported by advection. The Los Angeles basin is an area source of photochemical oxidants.
Axillary	occurring in the angle formed between any two plant organs.
Bifacial lesion	foliar injury that extends through the leaf.
Bifacial necrosis	death or collapse of tissue extending through the leaf.
Bio-indicator	plant species or varieties that are particularly sensitive to a specific pollutant so that they provide useful indicators of the presence of a particular pollutant.
Bleaching	complete loss of all pigmentation in leaf.
Bronzing	a brown discoloration that usually appears on the abaxial surface of leaves, and is often an advanced stage of the silvering or glazing typical of injury by PAN and other oxidents.
Chloride	the ionic form of the element chlorine.
Chlorosis	yellowing or paling of green parts of the plant due to loss of chlorophyll.
Chronic injury	injury which develops only after long-term or repeated exposure to an air pollutant, and expressed as chlorosis, bronzing, premature senescence, reduced growth, etc.; can include necrosis.
Clone	a group of organisms derived asexually from a single individual.

Coefficient of haze (COH)	a measurement of visibility based on the quantity of smoke and dust in 1,000 linear feet of air.
Conidium, conidia	spore-like asexual reproductive structure of certain fungi.
Conifer	plant that bears seeds in cones.
Cotyledons	the first leaves (1, 2, or more) of the embryo as found in the seed.
Cuticle, cutin	waxy protective layer covering primary plant parts.
Damage	a measure of the decrease in economic or aesthetic value resulting from plant injury by pollutants. (Term considered by some to be synonymous with injury).
Deciduous	characteristic of plants that shed leaves at the end of one growing season.
Defoliate	to cause a loss of foliage, especially prematurely.
Desiccation	drying out of foliage.
Dicot	having two cotyledon leaves.
Disease	an abnormal condition of the plant caused by a pathogen, or the environment.
Diurnal	occurring during the day; daily.
Dose	concentration of the pollutant multiplied by the duration of exposure.
Effluent	waste emitted to the environment.
Enation	growth of a stem or branch in a flattened rather than round condition.
Epidermis	the outer layer of cells on all primary plant parts.
Epinasty	leaf growing at right angle or downward to a stem rather than at an acute upward angle.
Fleck	collapse of a few cells in isolated areas in the upper layers of the leaf, resulting in tiny light-colored lesions.
Fluorosis	disease in cattle, sheep, or other grazing animals brought about by ingestion of fluorine-contaminated forage.
Fossil fuel	fuel derived from decayed organic matter from past geological ages, e.g., coal, oil.
Fungus, fungi	organisms that lack the ability to manufacture their own food but derive their nourishment by absorption from pre-existing organic sources.
Glazing	silvery appearance on the leaf.
Hypertrophy	refers most commonly to an abnormal enlargement.
Hypha, hyphae	thread-like filament consisting of vegetative body of some fungi.

Injury	any change in the appearance and/or function of a plant that is deleterious to the plant.
Inoculation	process involving introduction of disease-causing biological agent in tissues of the host plant.
Lapse rate	the rate of temperature change with elevation in the atmosphere.
Lesion	superficial injury or other circumscribed pathologic change in tissue.
Marginal	occurring along the periphery of the leaf.
Mesophyll	the inner ground tissue of a leaf consisting mostly of parenchyma cells.
spongy mesophyll	the loosely associated irregularly arranged parenchyma cells of the lower mesophyll of a leaf.
palisade mesophyll	layer of mesophyll cells oriented perpendicular to the upper epidermis of the leaf.
Monocot	having only one cotyledon leaf.
Mottle	degeneration of the chlorophyll in certain areas of the leaf giving the leaf a blotchy appearance.
Necrosis	death of tissue.
Palisade	layer of closely arranged mesophyll cells below upper epidermis of leaf.
PAN, peroxyacetyl nitrate	the principal constituent in a homologous series of compounds referred to as peroxyacyl nitrates or PAN's. Formed by photochemical reactions involving nitrogen dioxide and hydrocarbons.
Parenchyma	tissue composed of parenchyma cells.
Parenchyma cells	usually thin walled, vacuolated, and living, with varying shapes, but most commonly isodiametric.
Pathogen	any organism, usually fungus, bacteria, virus or parasite, or abiotic agent, capable of inciting disease.
Pathogenesis	process involving disease development.
Pesticide	any substance or mixture of substances intended for preventing, destroying, repelling or mitigating any insects, rodents, nematodes, fungi, weeds and other forms of plant or animal life or viruses, except viruses on or in – living man or other animals which are declared to be pests, and any substance or mixture of substances intended for use as a plant growth regulator, defoliant or desiccant. (PL 92-516, Federal Environmental Pesticide Control Act of 1972, Sec. 2 t,u).

Phloem	vascular tissue in plants that transports carbohydrates from leaves to roots.
Photochemical oxidants	pollutants resulting from the reaction of sunlight on automobile exhaust or other airborne gases.
Photosynthesis	the process carried on by green plants which utilizes light energy to split water, forming O_2 and chemical energy, which in turn is utilized to convert atmospheric CO_2 into carbohydrate.
Phytotoxicant	any agent that becomes toxic to plants.
ppb	parts by weight or volume of pollutants per billion parts by volume of air (usually refers to volume of pollutant if not so stated).
pphm	parts by weight or volume of pollutants per hundred million parts by volume of air (usually refers to volume of pollutant if not so stated).
ppm	parts by weight or volume of pollutants per million parts by volume of air (usually refers to volume of pollutant if not so stated).
Pustules	pimple-like bodies enclosing reproductive structures of some fungi.
Senescence	stage of development of plants or parts of plants encompassing the time from maturity to death.
Sepal	the outer, often leaf-like, whorl of a flower.
Stipple	dark-colored lesions on leaves resulting from pigmentation of injured cells.
Syndrome	a combination of symptoms and signs characteristic of an abnormal condition.
Synergism	the action of two independent factors resulting in an effect which is greater than the sum of the individual factors.
Temperature inversion	a condition in which temperature increases rather than decreases with altitude.
Urediospore	asexual reproductive structure of some fungi.
Vascular bundle	an organization of tissue in the plant for the transport of food, minerals, and water.
Xylem	vascular tissues in plants that transport water and minerals from roots to leaves.

INDEX

Abaxial layer, 2-10
Abiotic causal agents (of mimicking symptoms), 8-1
  construction damage, 8-4
  de-icing salts, 8-4
  lack or excess of water, 8-1
  nutrient excesses and deficiences, 8-3
  other pollutant-induced symptoms, 8-5
  pesticides, 8-4
  soil acidity, 8-3
  temperature – frost or cold injury, 8-2
  temperature – heat injury, 8-3
  winter injury, 8-2
Abscission, E-1
Accessory cells, 2-30
Adaxial layer, 2-10
Ammonia, 6-15
  acute injury, 6-16
  air quality standards, 6-17
  atmospheric chemistry, 6-15
  atypical symptoms, 6-16
  chronic injury, 6-16
  factors affecting plant response, 6-16
  injury threshold, 6-17
  monitoring data, 6-15
  relative sensitivity, 6-17
  sources, 6-15
  symptomatology, 6-16
Apical meristem, 2-5, 2-7
Assessment of damages, 1-4
Axillary buds, 2-6

Bark, 2-6
Biotic causal agents (of mimicking symptoms), 8-5
  bacteria, 8-6
  diseases, 8-5
  fungi, 8-5
  insects, 8-6
  nematodes, 8-6
  viruses and mycoplasmas, 8-6
Blade, 2-8
Bullheading, 6-12
Bulliform cells, 2-28
Bundle sheath, 2-11

Cadmium, 6-24
Carbon monoxide, 6-21
  air quality standards, 6-21
  sources, 6-21
  symptomatology, 6-21
Cells, 2-1
Cell wall, 2-1
  primary, 2-1
  secondary, 2-1
Chlorine, 6-2
  acute injury, 6-3
  air quality standards, 6-4
  atmospheric chemistry, 6-2
  chronic injury, 6-3
  factors affecting plant response, 6-4
  leaf tissue analysis, 6-5
  mimicking symptoms, 6-3
  monitoring data, 6-3
  relative sensitivity, 6-4
  sources, 6-2
  symptomatology, 6-3
    broad-leaved plants, 6-3
    coniferous plants, 6-3
    narrow-leaved plants, 6-3
  threshold concentrations, 6-4
Chlorophyll, 2-2
Chloroplast, 2-2
"Christmas tree" pattern of chlorosis, 5-5
Chromatin, 2-2
Chromosomes, 2-2
Copper, 6-24
Cork cambium, 2-6
Cuticle, 2-10
Cytoplasm, 2-2

Darmous, 5-1
Diagnosis, 10-1
  basic procedure, 10-1
  systematic approach, 10-5
Diagnostic routine, 10-8
Diurnal temperature variations, 9-6
Dry adiabatic lapse rate, 9-3

Ecological index number, D-1
Emergence tipburn of eastern white pine, 3-10
Emission inventory, 9-2

Endodermis, 2-4
Environmental lapse rate, 9-3
Epidermis, 2-4
Epiphytes, D-1
Ethylene, 6-10
  acute effects, 6-12
  air quality standards, 6-13
  atmospheric chemistry, 6-10
  chronic effects, 6-12
  factors affecting plant response, 6-13
  leaf tissue analysis, 6-14
  monitoring data, 6-11
  relative sensitivity, 6-14
  sources, 6-10
  symptomatology, 6-11
  traumatically induced, 6-11

Flowers, 2-12
Fluoride, 5-1
  air quality standards, 5-17
  atmospheric chemistry, 5-3
  chemical analysis, D-4
  factors affecting plant response, 5-10
  injury threshold, 5-13
  leaf tissue analysis, 5-17
  monitoring data, 5-4
  sources, 5-1
  symptomatology, 5-4
    broad-leaved plants, 5-5
    coniferous plants, 5-9
    fruit, 5-10
    narrow-leaved plants, 5-7
Fluorine, 5-1
Fluorspar, 5-1
Frequency – coverage number, D-1

Grape stipple, 3-10
Guard cells, 2-9
Guttation, 2-10

Heavy metals (see also Lead – Cadmium – Copper – Mercury vapors), 6-24
Herbicides, 6-29
Hydathode, 2-10
Hydrogen chloride, 6-6
  acute injury, 6-7
  air quality standards, 6-8
Hydrogen chloride (continued)
  atmospheric chemistry, 6-6
  chronic injury, 6-7
  factors affecting plant response, 6-7
  injury threshold, 6-8
  mimicking symptoms, 6-7
  monitoring data, 6-7
  relative sensitivity, 6-8
  sources, 6-6
  symptomatology, 6-7
    broad-leaved plants, 6-7
    coniferous plants, 6-7
    narrow-leaved plants, 6-7
Hydrogen fluoride, 5-1
Hydrogen sulfide, 6-18
  acute injury, 6-19
  air quality standards, 6-19
  atmospheric chemistry, 6-18
  chronic injury, 6-19
  factors affecting plant response, 6-19
  injury threshold, 6-19
  leaf tissue analysis, 6-20
  monitoring data, 6-18
  relative sensitivity, 6-19
  sources, 6-18
  symptomatology, 6-19

Index of atmospheric purity (IAP), D-1
Interactions (see Pollutant – pollutant interactions – Pollutant – pathogen interactions)
Intercalary meristem, 2-7
Internode, 2-6

Lamina, 2-8
Lapse rates, 9-3
Lead, 6-23
  effects on plants, 6-23
  sources, 6-23
Leaf arrangement, 2-8
  alternate, 2-8
  opposite, 2-8
  whorled, 2-8
Leaf function, 2-9
Leaf growth, 2-11
Leaf sheath, 2-8
Leaf structure, 2-10
Leaf types, 2-10
  broadleaf, 2-10

Leaf types (continued)
  compound, 2-8
  narrowleaf, 2-10
  needles, 2-10
  simple, 2-8
Legume, 2-5
Lenticel, 2-6
Lichen, D-1

Median effective dose, 1-6
Median toxic dose, 1-6
Mercury vapors, 6-24
  relative sensitivity, 6-25
  sources, 6-25
  symptomatology, 6-25
Mesophyll, 2-10
Meteorological aspects of air pollution, 9-2
  atmospheric stability and temperature, 9-3
  wind speed and direction, 9-2
Middle lamella, 2-1
Mimicking symptoms (see Abiotic causal agents, Biotic causal agents)
Mitochondrion, 2-3

Nitrogen dioxide (see NO_x)
Node, 2-6
Nodule, 2-5
NO_x, 3-22
  air quality standards, 3-29
  atmospheric chemistry, 3-22
  factors affecting plant response, 3-27
    external, 3-27
    internal, 3-28
  injury threshold doses, 3-28
  leaf tissue analysis, 3-31
  monitoring data, 3-23
  relative sensitivity, 3-29
  sources, 3-22
  symptomatology, 3-26
    broad-leaved plants, 3-26
    coniferous plants, 3-27
    narrow-leaved plants, 3-27
Nuclear envelope, 2-2
Nucleus, 2-2

O'Gara, 4-8
Onion tipburn or blight, 3-10
Oxides of nitrogen (see NO_x)
Ozone, 3-1
  air quality standards, 3-13
  factors affecting plant response, 3-11
  injury threshold doses, 3-12
  leaf tissue analysis, 3-16
  relative sensitivity, 3-14
  symptomatology, 3-8
    broad-leaved plants, 3-8
    coniferous plants, 3-9
    narrow-leaved plants, 3-8
Ozone needle mottle of Ponderosa Pine, 3-11

Palisade layer, 2-10
PAN (peroxyacetyl nitrate), 3-17
  air quality standards, 3-19
  factors affecting plant response, 3-19
  injury threshold doses, 3-19
  leaf tissue analysis, 3-21
  mimicking symptoms, 3-18
  relative sensitivity, 3-19
  symptomatology, 3-17
    broad-leaved plants, 3-17
    coniferous plants, 3-18
    narrow-leaved plants, 3-18
Particulate matter, 6-27
Peroxyacetyl nitrate (see PAN)
Pesticides, 6-29
  leaf tissue analysis, 6-30
  relative sensitivity, 6-30
  sources, 6-29
  symptomatology, 6-30
Petiole, 2-8
Phloem, 2-4
Photochemical oxidant, 3-1
Photochemical reaction, 3-2
Photosynthesis, 2-2
Pith, 2-6
Plasmalemma, 2-1
Plastid, 2-2
Pollutant – pathogen interactions, 7-5
Pollutant – pollutant interactions, 7-1
  sulfur dioxide and hydrogen fluoride, 7-4
  sulfur dioxide and ozone, 7-2
  sulfur dioxide and nitrogen dioxide, 7-2

Ray, 2-6
Resin duct, 2-6
Rhizome, 2-7
Roholm, 5-1
Root cap, 2-5
Root growth, 2-5
Root types, 2-4
  adventitious, 2-4
  fascicled, 2-4
  fibrous, 2-4
  tap, 2-4
Runner, 2-7

Silicone tetrafluoride, 5-4
Sleepiness, 6-12
Smog, 3-1
  photochemical, 3-1
Sources of air pollution, 9-1
  industrial sources, 9-1
  mobile sources, 9-2
  rural sources, 9-2
  urban sources, 9-2
Spongy layer, 2-10
Stem growth, 2-7
Stem types, 2-6
  gymnosperm, 2-6
  herbaceous dicot, 2-6
  monocot, 2-6
  woody dicot, 2-6
Stolon, 2-7
Stomata, 2-8
Sulfate ion, 4-4
Sulfite ion, 4-4
Sulfur dioxide, 4-1
  air quality standards, 4-10
  atmospheric chemistry, 4-2
  factors affecting plant response, 4-6
  leaf tissue analysis, 4-11
  monitoring data, 4-2
  relative sensitivity, 4-10
  sources, 4-1
  symptomatology, 4-4
    broad-leaved plants, 4-5
    coniferous plants, 4-6
    narrow-leaved plants, 4-6
Sulfuric acid mist, 4-2
Suture red spot, 5-10
Synergism, 7-1

Teratogenic causal agents (of mimicking symptoms), 8-7
  genetic disorders, 8-7
Temperature inversion, 9-3
Tonoplast, 2-2
Topographical effects on dispersion of pollutants, 9-7
Transpiration, 2-9
2,4-D; 2,4,5-T; 2,4,5-TP, 6-30
  relative sensitivity, 6-30

Vacuole, 2-2
Vascular cambium, 2-5
Viruses and mycoplasmas, 8-6

Weather fleck of tobacco, 3-10
Wood, 2-6

Xylem, 2-4

Zinc, 6-24